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VETERINARY MEDICINE

NOVEMBER 1943

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In experiments in England, penicillin was found so effective that within five days the streptococci was destroyed in 76% of 54 burns and scalds in various stages of healing.

By extracting carotene oil from a ton of dehydrated alfalfa leaf meal, it has been found by Michigan State College, that an amount equivalent to 75 pounds of fish liver oil concentrate (10,000 units of vitamin A and 500 units of vitamin D), can be secured.

That tires wear out as much as four times as fast at 65 as at 35 m.p.h. and that speed is the most important factor in determining the rate of tire wear has been determined in a study conducted by the Engineering Experiment Station of Iowa State College and the Public Roads Administration. On an average, tire mileage at 65 m.p.h. is 18,700 while at a maximum speed of 25 m.p.h. it is 69,500.

It has been found by the Mellon Institute that a very fine clay called bentonite when added to distillery wastes attracts the protein molecules and forms a precipitated mass which may be filtered off or centrifuged. This is a comparatively cheap and simple method by which to recover these valuable distillery waste proteins. It is thought that the stock feeding value of the protein so recovered is unimpaired except for increase in ash content.

Dry ice, as well as regular icing, is now required on some army shipments of fresh meat and frozen products when they are transported through areas of high summer temperatures or where they may be delayed in transit.

It has been suggested that impaction of the epiglottis may be the explanation of sudden death from blast. If it is proved that such is the case, death can be averted if the epiglottis is promptly replaced and/or if the vacuum holding it down is abolished by plunging a hollow needle into the trachea. Artificial respiration is futile until this is accomplished.

Experiments of the U. S. Department of Agriculture indicate that sweet potato meal, made by dehydration, is a promising substitute for corn in steer feeding as it is about 3% more efficient than cracked shelled corn. But recent feeding trials conducted by the Louisiana Experiment Station, indicate that sweet-potato meal has 95% of the feeding value of corn. Since the amount of nutrient produced on an acre of sweet potatoes is four times the amount of that of corn raised on the same acreage, the discovery of the high stock feeding value of sweet potatoes should be of great interest in the South where there is much more land planted to the sweet potato than to corn.

Large hogs not only have more fat but the fat yields more lard than fat of smaller pigs, according to report of experiments of the B.A.I.

Pullorum Disease Reduced

Since official testing for pullorum disease of poultry breeding stock began in 1936 the disease has been reduced by one-third. This was done on a Federal-State basis.

Karl Landsteiner, a member of the Rockefeller Institute for Medical Research, died June 26, 1943. He was most widely known for his demonstration that there are a certain number of types of blood and that a successful transfusion (whole blood) demands that the recipient receive his identical type. Landsteiner was also the first man to transmit poliomyelitis to monkeys—thus making experimental studies of the disease possible.

Cash received for livestock represents American farmers' largest source of cash income. During the last full calendar year livestock growers received nearly one-third of their income, or a total of $4,811,000,000 for their livestock. The sum is $1,569,000,000 more than they received for meat animals during the preceding year and the rising trend has continued in 1943. The increase —approximately 48%—reflects not only a higher trend of livestock prices but also material larger marketings.

Vitamin C of Milk Retained

When evaporated milk is being canned if the sealing is done in an atmosphere of nitrogen or in vacuum, the vitamin C content after six months storage shows a 50% increase over that of evaporated milk canned in contact with the air and stored for an equal length of time.

The process for keeping and sealing in the vitamin C was developed by investigators in the Dairy Department of the Pennsylvania State College. When canning the milk as described it may be fortified with more of the vitamin and thus made capable of supplying a large part of the essential vitamin C of a well balanced diet.

Anthrax in Minks

In September an outbreak of anthrax occurred on several mink ranches near Marathon, Wisconsin. Before the disease was diagnosed 288 minks had died.

Meat from a cow, which in the morning had seemed well but in the afternoon had died suddenly, was fed on all of the ranches where the disease was found.

Diagnosis of anthrax was made by Doctor Wisnicky to whom the mink carcasses had been sent. Laboratory examination of the specimens confirmed the diagnosis.

★ ★ ★ BUY WAR ★ ★ ★
STAMPS AND BONDS

Of two cases of Foot-and-mouth disease reported in the British Medical Journal (Feb., 1943) one was acquired from a splinter from a butcher's block and the second from raw milk. Diagnosis of the first case led to an investigation and the finding of the disease among cattle in the district. The possibility, that because of the comparative mildness of foot-and-mouth disease in man, the disease may be frequently overlooked and he may become a carrier and be the means of spreading the disease among cattle, should be considered.

Bacillus pestis Retains Virulence Many Years

That *Bacillus pestis* retained its viability and virulence for full 20 years on beef infusion agar stored at 10°C. and without transfer, has just been announced[1] by the U. S. Public Health Service.

Of the 48 tubes, cultured and stored in 1923, when subcultured on horse meat infusion agar, 33 showed growth in 2 to 7 days. The growth from the 33 positive tubes were each injected into a guinea pig. Of six of the pigs which were dying and three which were already dead at the end of the first week after subcutaneous injection of the bacillus, all showed typical gross lesions of acute bubonic plague and there were great numbers of *B. pestis* in the smears of spleen and glands—20 years after storage.

[1] Francis, Edward, 1943. Twenty-year survival of virulent *Bacillus pestis* without transfers. *Pub. Hlth. Rpt.*, 58:37, pp. 1379-1382.

A Deterrent

August Hamm, 75, was promptly sentenced to death by a special court at Darmstadt, Germany, for illegal livestock slaughter and black market trading, according to a report from Berne, Switzerland.

In addition to furnishing the essential proteins, meats are important sources of minerals, thiamin, niacin and riboflavin. Meats are more nutritious when served rare than when thoroughly cooked. Overcooking causes a loss of vitamins, but is necessary in the case of pork as a safeguard against trichinosis.

The Wisconsin Agricultural Experiment Station has combined soybean oil meal and infertile eggs to make a cheap protein supplement for chick rations—poultry receiving this food gained nearly as fast as chickens which were fed a standard feed. To prepare the feed the eggs are hardboiled, run through a food chopper (shells and all) and enough soybean oil meal is added to prevent stickiness.

Swine Disease Identified as Vesicular Stomatitis

A recent outbreak of a disease in hogs, having symptoms closely resembling those of both vesicular exanthema and the foreign plague, foot-and-mouth disease, proved to be vesicular stomatitis, the U. S. Department of Agriculture reports. Characterized by sore feet and blisters on the snouts, the malady appeared during August among hogs in an anti-hog cholera serum-producing plant in Kansas City, Kans.

Vesicular stomatitis affects mainly horses and sometimes cattle, but no previous natural cases of the disease in swine are on record, though research workers have been able to produce lesions of the malady experimentally in swine.

In the recent outbreak, the origin of which is unknown, the first symptoms were high temperature and lameness. Blisters soon appeared involving usually the feet but in some cases the snouts also. Since these same symptoms are indications of both foot-and-mouth disease and vesicular exanthema Drs. H. W. Schoening and A. B. Crawford, went from Washington by plane and made a series of tests with several species of animals of varying susceptibility to the suspected types of virus. Horses, which are resistant to the virus of foot-and-mouth disease, readily developed tongue lesions, when inoculated artificially with the virus of the strange swine disease. That observation and others, together with negative results from contact exposure of cattle to the infected swine, ruled out foot-and-mouth disease, which has been nonexistent in the United States since 1929.

Other tests involving cattle, swine and guinea pigs narrowed the diagnosis down to vesicular stomatitis, notwithstanding the rarity of this disease in swine.

The Effect of External Temperature on Shock

In a series of experiments* involving the use of rats, guinea pigs and dogs in which shock had been produced experimentally, the author found that external temperature near that of normal body temperature produced longer survival time.

The state of shock was produced in anesthetized animals by intestinal stripping or trauma to appendages. The external temperature was regulated by placing the shocked animals on either ice bags or hot water bottles.

It was found that neither temperature extremes produced favorable results. Furthermore, it was pointed out that the application of external heat, as recommended by many first aid manuals, works against the efforts of nature to save the shocked individual. The peripheral vasodilation caused by heat defeats the effects of the reflex peripheral vasoconstriction which is the natural adaptation for the decrease in blood volume.

In summarizing, it was concluded that the external application of either heat or cold considerably shortened the life of the animals. The optimum room or environment temperature was found to be near the physiological temperature of animal involved.—J. E. G.

*Wakim, K. G., and Gatch, W. D., 1943. The effect of external temperature on shock; an experimental study. *Jnl. A.M.A.*, 121:12, pp. 903-907.

Examination of Thoroughbreds*

By M. J. O'ROURKE

"DOC," the old-time trainer used to say, "this horse is as solid as a bell of brass. There's not a pimple on him."

Before the advent of legalized horse-racing, the old-time trainer's word was usually good enough. If he said a horse was solid, the horse ran—out of the money, perhaps, but he ran. If the trainer said his horse was sick or lame, the horse was scratched. Those who had paid their money to see the animal perform were assumed to have no privileges or interest in the matter.

Today the Racing Commission of the various states where horse-racing is a legalized and highly profitable sport have provided rules and regulations governing which horses may and which may not run and also the conduct of the owners, trainers and riders. Chief among these rules is the requirement that a thorough examination of the animal be made by a licensed, practicing, official veterinarian.

Before the commissions inspired this rule, examinations were more or less perfunctory. The only horses examined were the horses which the owners or trainers wanted scratched. Often the "sickness" or "lameness" was only a realization that the mount was out of his class. If competition turned out to be inferior, the same owners had a "running fool" who was "sound as a bell of brass"! "Sound" horses were examined as they entered the paddock just previous to the race. Obviously, a practical and thorough examination under such conditions was impossible.

When racing commissions were first appointed, they recognized this lack of examination as one of the principal rallying points for the enemies of racing, and immediately insisted upon examination of *all* horses in every race.

In California, the infant commission instituted an examining procedure which I believe to be the best protection against skullduggery and trickery ever given the public—before or since.

Horses on which owners put in "scratch claims" were examined by the veterinarian just as they had been previously. But the important feature of California's system was the adoption and maintenance of the "receiving barn." Each horse, racing on a given day, was required to be in the receiving barn one hour before his race.

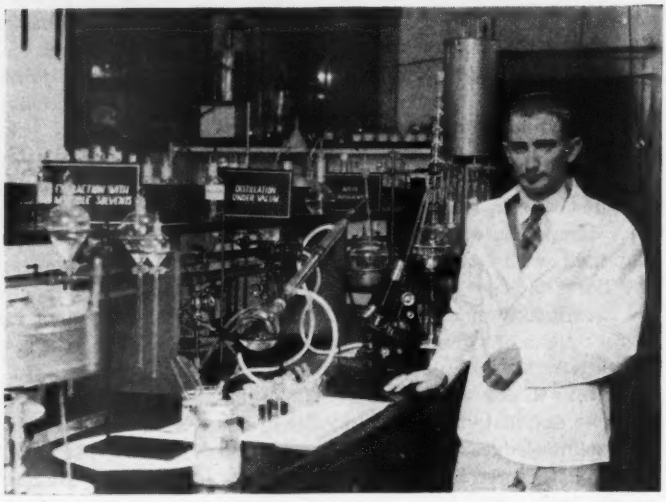
As each horse was on his way to the barn, he was observed by the track veterinarian and his assistant. A more thorough examination was made while the animal was in

the receiving barn. Throughout the entire "quarantine period," the horse was accompanied by his groom who was responsible for the well-being of the animal while in the barn.

In the paddock, a veterinarian observed the approach of the horses from the barn. He watched them as they were saddled and walked previous to entering the track for the race. This careful observance served a dual purpose. A horse may be a border-line case; his soreness or lameness may not be such as to warrant an opinion that he is not "serviceably sound." Such cases were given critical inspection. When the jockey, with his weight up, is placed on a horse's back, defects will show themselves before the animal has traveled very far on his way to the starting post. The veterinarian

*Doctor O'Rourke died suddenly at Los Angeles, June 24, 1943, while the California State Board of Veterinary Examiners, of which he was a member, was conducting an examination. He was a past president of the California Veterinary Medical Association and of the Bay Counties Veterinary Association and a former president of the San Francisco Veterinary College. At the time of his death, he was chairman of the California Procurement and Assignment Service for Veterinarians under the War Manpower Commission. This address is believed to have been his last before any group of veterinarians.

then called this horse to the attention of the stewards and if, in their judgment, the condition warranted it the horse was scratched. Contrary to the opinion of certain professional character-assassins, track stewards, as a whole, are honest, high-class men with a desire to do their part to keep racing clean. I can vouch personally for the stewards of Tanforan and Bay Meadows tracks and I believe that officials of other tracks are equally high-minded. I have never had a horse allowed to race by the stewards when my report indicated that the animal was not in condition to race because of illness or lameness.



The laboratory at the State College of Washington where pioneer work in saliva testing was done about a decade ago. O. B. McRory (pictured) made many of the tests which were done under the supervision of the veterinary department of the college

After the race is run, the judges or stewards select two horses, usually the winner and the second or third runners, to receive the saliva test, given by the Commission's veterinarian. This test ascertains whether or not a stimulant or drug of any kind has been administered. The collection of saliva is made in the presence of the owner or trainer of the horse and he signs as a witness. The jars containing the saliva are then sealed and sent to the Commission's chemist for analysis.

With the advent of the receiving barn, the time element made it practically impossible to employ, illegally, drugs which retain their efficiency for a limited time only. From the moment the horse left his own stable, 10 or 15 minutes elapsed before he reached the receiving barn. He spent an hour inside the barn, 20 minutes in the paddock before the race and 10 minutes in traveling from paddock to the starting-gate. Constant inspection and supervision were exercised during the entire hour and forty minutes occupied by this procedure.

Although the present method lacks the almost fool-proof system of the receiving barn, it has a number of good points. During the morning, the horses are examined by two veterinarians and two assistants. A slip is signed by the groom in charge of each animal, accepting responsibility for the animal until post time. Further examination is made in the paddock. After the race the Commission's veterinarian makes not only the saliva test, but the urine test as well.

The weakest link in this procedure is the regulation requiring the groom to stay with his horse until post-time. This is a pretty stringent requirement, covering as it does a long period—sometimes as much as eight hours. To secure enforcement of this rule would require a considerable police department. Obviously it would be easily possible for an unscrupulous owner or trainer to get rid of his groom for a time, on one pretext or another.

The public has other protections. Each horse is accompanied, when he arrives at the track, by a copy of his certificate of

registration, the original of which was filed with the Jockey Club when the horse was foaled. The animal's markings are closely checked and when the identifier is satisfied that they agree with those described in minute details on the certificate or shown in pictures, the horse is admitted to the track. This identification report follows the horse during his entire life-time—no criminal ever had a more complete dossier. By means of it, the once common "ringer" has become a thing of the past in racing.

The original examination of the horse then proceeds from identity to physical condition. Pulse and heart action are taken. Conjunctiva and nasal passages are examined, temperature is taken and legs are examined while the animal is standing. The horse is jogged or trotted away from the examiner then back toward him, to determine signs of soreness. The veterinarian's report is written in duplicate, the original going into the files of the Racing Commission, the duplicate is retained by the groom who acknowledges both copies by his signature. The horse is then in charge of the groom. It is upon him that responsibility for unexplained "sudden illnesses" will be placed.

Good thoroughbreds come in all sizes and shapes. Characteristic of the first class thoroughbred, however, are the sloping shoulders, short canons and springy postern. This build is necessary to withstand the great strain of racing. Generally speaking, the thoroughbred of this confirmation has courage, soundness and that vague quality we call "intestinal fortitude." He is also less subject to the many types of lameness common to race horses.

Probably the seat of most lameness is the tendons and joints of the fore limbs. This is readily understood when we take into consideration the fact that they are the pillars which absorb the shock; the hind legs are the propellers. Foot lameness affects a great number of animals. Less common, but sometimes equally serious, is shoulder lameness.

Obscure lamenesses are sometimes caused by intestinal parasites. I recall one case in particular where a first-class thoroughbred by that wonderful sire, Equipoise, was being constantly refused permission to race both by my associate at Bay Meadows and myself; the reason being a soreness manifested whenever the horse was jogged. This horse had been fired and blistered on both knees and later on one fetlock. He had also had a tour with "the green grass doctor," dependable old dame Nature, who cures a great many cases of soreness and lameness

Best American Races 100 Years Ago

BEST RACES AT THREE-MILE HEATS.

Name.	Sire.	Age. Yrs.	W'ght. lbs.	Time.	Beating the undermentioned Horses.	Course.	Date.
Blue Dick.....	Imp. Margrave	5	114	5.44—5.38½	Heiress, and Young Dove.....	Trenton, N. J....	May, 1842.
Passenger	Langar.....	3	90	3d heat 5.44	Argyle, and others.....	Do.	
Sally Walker....	Timoleon	5	107	5.44—5.42	Ariel	Broad Rock, Va...	Sept. 1827.
Andrewetta.....	Andrew.....	5	111	5.48—5.42½	Bandit, and Raritan.....	Trenton, N. J....	May, 1840.
Omega.....	Timoleon	5	111	5.48—5.43—5.47 ..	Clarion, and Truxtun.....	Do.	Oct. 1839.
Sarah Bladen ...	Imp. Leviathan	6	107	5.49—5.41½	Lucy Fuller	New Orleans.....	March, 1840.
Master Henry...	Henry	6	118	5.47½—5.40—5.56 6.01	Wonder (who won 1st heat), Argyle (who won 2d heat), and others.....	Kendall's, Md.....	May, 1839.
Sussex	Sir Charles ...	4	100	5.46—5.43	Polly Hopkins, Sally Hornet, May Day, and others.....	Broad Rock, Va...	Spring, 1830.
Arbaces.....	Bertrand	4	86	5.45—5.43	Naked Truth, and Alice Riggs.....	St. Francisville, La.	April, 1837.
Clara Howard...	Imp. Barefoot .	4	101	5.47—5.45	Henry Archy, and Charles Kemble.....	Union Course, L. I.	June, 1835.
George Martin ..	Zinganee	5	100	5.40—5.46	Tom Marshall, Lix Hewitt, and Humming Bird	New Orleans.....	March, 1842.
Treasurer	Imp. Roman ..	4	104	5.42	Andrewetta.....	Union Course, L. I.	June, 1840.
Black Maria....	Eclipse	7	123	5.42	De Witt Clinton	Po'keepsie, N. Y.	Oct. 1833.
Red Bill.....	Medoc	4	100	5.40—5.48—5.49 ..	Blacknose (who won 1st heat), Occident, and others.....	Lexington, Ky....	May, 1840.
Astor	Ivanhoe	5	110	5.45—5.44	Telemachus, Hector Bell, Rienzi, and others.	Washington, D. C.	June, 1841.
Pressure	Trumpeter ...	4	86	5.49—5.44	Dick Chinn, Pollard, Pete Whetstone, and Susan Yaudell	New Orleans.....	April, 1838.
Belle Peyton....	Andrew	4	100	5.45—5.44	Stockton, Mary Selden, Red Fox, and Rowena	Kendall's, Md....	May, 1838.
Blacknose	Medoc	4	100	5.45—5.46	Laura Webster.....	Lexington, Ky....	Sept. 1840.
Ripple	Medoc	4	100	5.51—5.47—5.44—5.52	Lucy Fuller, Maria Collier, Laura Webster, and Collier	Louisville, Ky. ...	Oct. 1840.
Bertrand	Sir Archy	5	112	5.47-5.48-5.53-5.54	Aratus, and Creeping Kate.....	Charleston, S. C...	Feb. 1826.
SarahWashington	Zinganee	5	107	5.40—5.45	Milton Brown, and others.....	Kendall's, Md....	May, 1842.
Flirtilla	Sir Archy	5	113	5.59—5.42½	Ariel, in a match	Union Course, L. I.	Oct. 1833.
Black Maria....	Eclipse	7	123	6.02—6.07—5.48..	Virginia Taylor, Rolla, Alice Grey, & Jackson	Do.	May, 1833.
Blue Dick.....	Imp. Margrave	5	114	5.49—5.39½	Sarah	Alexandria.....	June, 1842.

for which veterinarians get full credit.

Despite these measures, the horse still showed soreness. My associate, Dr. O. A. Longley, shared with me a theory concerning the possible cause of this type of lameness. We were not consulted by the veterinarian in charge of the case, and we were further constrained by the fact that we do not practice during the time we are employed at the track. However, one day the owner of the horse, honestly believing that the lameness had subsided, entered his mount in a race. Examination showed lameness. The horse was scratched. In desperation he turned to me and said:

"For heaven's sake, Doc, haven't you any idea about this? My veterinarian has tried everything, but without benefit"!

"Doctor Longley and I have discussed this case," I told him, "and we do have a theory. Our suggestion is that analyses be made of some of the animals feces and blood." When these were made by Dr. R. Northrup of San Francisco, a heavy infestation of parasites was found.

The sequel was further treatment by the veterinarian in charge of the case, rapid improvement and, finally, a winning race at an Eastern track. It constituted another reminder of the possibilities of this type of "lameness" whenever I was called upon to treat a horse in which, after careful thorough examination, no symptoms could be found.

One more point about obscure lameness which appeared to crystallize itself in my experience was the rarity of thrombosis as a cause. Another type of lameness which requires extremely careful examination is the incipient quartercrack. The history here is that the animal "came out of the race" or "workout" all right but showed some lameness the following morning. The severity of this particular lameness increases with the rapidity of the gait—the lameness being most pronounced when the foot bears the greatest weight. Examination will reveal considerable heat at the "coronary band," more pronounced in the outside quarter; feeling the wall carefully just below the band will give a sensation of roughness slightly different from that of the inside quarter. The crack had not been manifested earlier because the trauma is below the periople but invariably a crack is readily discernible the next day, especially if the foot is forced to bear weight. Generally speaking, contracted heels and atrophy of the frog are present when either sand crack or quartercrack results.

BEST RACES AT FOUR-MILE HEATS.

Name.	Sire.	Age. Yrs.	W'ght. lbs.	Time.	Beating the undermentioned Horses.	Course.	Date.
Fashion	Imp. Trustee	5	111	7.32½—7.45	Boston, in a match, [he 9 yrs. old, with 126 lbs.]	Union Course, L. I.	May, 1842.
Eclipse	Duroc	9	126	7.37—7.49—8.24	Henry, in a match	Do.	May, 1823.
Boston	Timoleon	5	114	7.40	Charles Carter, who broke down end of 3d mile.	Do.	June, 1838.
Wagner	Sir Charles	5	110	7.48—7.44	Grey Eagle, Queen Mary, and Hawk-Eye	Louisville, Ky.	Sept. 1839.
Wagner	Sir Charles	5	110	7.51—7.43	Grey Eagle, (five days after preceding race)	Do.	Oct. 1839.
Grey Medoc	Medoc	6	110	7.35—8.19—7.42 8.17	Altorf (by Imp. Fylde), and Imp. Denizen	New Orleans	March, 1841.
Lady Clifden	Sussex	4	101	7.44 — 7.43½ — 7.56½	Fanny Wyatt, Picton (by Imp. Luzboro'), and Mingo	Union Course, L. I.	Oct. 1837.
Jim Bell	Frank	4	86	7.37—7.40	Sarah Bladen, and Martha Carter	New Orleans	March, 1842.
Miss Foote	Imp. Consol	4	83	8.02—7.35	Earl of Margave, Hannah Harris, and Luda	Do.	March, 1842.
Sarah Bladen	Imp. Leviathan	7	115	7.45—7.40	Luda, Imp. Esper Sykes, and Westwind (by Imp. Chateau Margaux)	Do.	March, 1841.
Omega	Timoleon	4	97	7.40—8.01	Mediator, Medora, Prince George, and Wonder	Washington, D. C.	Oct. 1838.
Duane	Imp. Hedgeford	4	100	7.42½—7.58½	Belie Peyton (who won 1st heat), and Stockton	Baltimore, Md.	May, 1838.
Fanny Wyatt	Sir Charles	3	87	7.48—7.53	Mingo	Trenton, N. J.	Nov. 1836.
John Bascombe	Bertrand	5	114	7.49—7.51	Post Boy, in a match, (course heavy)	Union Course, L. I.	June, 1836.
Eutaw	Imported Chateau Margaux	5	110	8.01—7.43	Sarah Washington, Bengal, Pryor, and others.	Washington	May, 1842.
Miss Foote	Imp. Consol	4	97	7.42—7.40	Argentile, Alice, Corneal, Kate Holton, and Lavota	Lexington, Ky.	Sept. 1842.
Miss Foote		4	97	7.36½-7.39-7.51½	George Martin	New Orleans	Dec. 1842.

The racing tables given on this and the preceding page, from Youatt's "The Horse" (1843), are interesting chiefly for the long distances and inferentially the hardihood of the animals. The record of trotters of that day is even more astounding in that heats were often five miles, and as many as four were run in an afternoon. Further, private races were commonly 10 miles in length and often up to 20 miles, while occasional races of 30, 50, and even 100 miles were run. Thus "Topgallant" trotted 12 miles in 38 minutes; Yankee Sal 15 miles on a heavy course in 48 minutes; Jerry, 17 miles in 58 minutes; Paul Pry, 18 miles in 59 minutes; Chancellor, 32 miles in less than two hours, and Tom Thumb, 100 miles in 10 hours and 7 minutes. One hundred years of selective breeding have increased the speed of our race horses materially, but 100 years of feeding on timothy hay and oats have reduced the stamina to an even greater degree.

Contraction of the foot is common in the thoroughbred and may not be detected in the early stages. It may go unnoticed until the frog is atrophied. In these cases, the gait is uncertain. Stumbling is a common symptom. Such cases should always be examined carefully from behind, to avoid mistaking this case for shoulder lameness or navicular disease. The horse-shoer is sometimes used to "manufacture" a crack to fool the examining veterinarian. This deception is practiced by "opening the heels," thus giving the appearance of the heels being wide apart, and the horse is shod accordingly. The lameness is controlled temporarily by standing the horse many hours in ice packs or by having a pseudo-veterinarian "doctor" the plantar nerve. This type of trickery is one of the best arguments for the receiving barn, because it is one in which the time element most often proves fatal to the dishonest trainer. There are many theories as to the cause of contraction of the foot. I am inclined to believe that while heredity and faulty diet during the early development, defective blood supply and loss of tissue vigor, may be contributing factors, concussion is the immediate cause. The foot of the thoroughbred is usually strong, round, short-toed, open at the heels and has a prominent frog. When the frog does not perform its normal resilient function and fails to absorb any of the concussion the pain is intensified.

Navicular disease represents at the outset an equally difficult problem. There is probably no other type of lameness so difficult to diagnose in its early stages. Pointing of the affected limb is usually an early sign, but it is not conclusive because the same symptom occurs in nearly every type of lameness within the horny box. Navicular lameness is intermittent and slight, very slow in its onset. This gradual progress further complicates diagnosis. When the animal is taken out of its stall it moves stiffly and the veterinarian suspects lameness. But palpation does not reveal any heat or tenderness in the leg or foot and he may tentatively diagnose the stiffness as a slight strain. Later on, not even a slight sign or pain or stiffness appears. The horse is taken for a "workout" and the exercise boy reports that the animal, at times, acted as if he were afraid to exert himself. At this point, the horseman or pseudo-veterinarian usually decides that the shoulder should be steamed out or blistered, and the horse rested for a couple weeks. Unfortunately, the same symptoms, now probably more clearly defined, recur when the horse returns to training. At this stage, there is

A young thoroughbred broodmare raised at the Front Royal Remount Depot and her first foal, a great-granddaughter of Man O' War

an inclination to go on the toes and the horse's movement becomes stilty and rather uncertain. The veterinarian's diagnosis is still tentative.

Later the symptoms increase in severity and changes in the hoof take place. The limb is pointed or advanced. Pointing with the heel elevated is a very common sign, no doubt to relieve pressure from the perforans tendon. The veterinarian may corroborate his tentative diagnosis by placing the toe of the affected foot on a small block of wood and, when the opposite limb is lifted, the horse will manifest severe pain. Later the lameness is very severe and more or less continuous. There is no other form of lameness in which the pain is so pronounced (out of all proportion to the heat of the foot or tenderness of the adjacent parts) as it is in navicular arthritis. The hoof is more or less upright. The heels contract. The frog, at first protruding and hard, gradually becomes atrophied. The animal constantly assumes postures intended to save its heels. It is easy to mistake navicular disease for contracted foot. This incorrect diagnosis may lead to shoeing, to give the frog pressure. This device will prove beneficial in cases of contracted foot, but will only aggravate the lameness caused by navicular disease.

There are methods of differentiating between the two. Extreme flexion or extreme extension of the joint is one useful method; in contracted heels, there will be no increase of pain, but in navicular disease the pain is intensified. Sesamoiditis is also easily mistaken for navicular disease. The symptoms are identical in many cases—pointing, maintaining the leg in a state of flexion, walking feelingly and uncertainly. However, on palpation, local symptoms in the fetlock joint, such as heat and pain and slight swelling. are found in sesamoiditis.

Among lesser important types of lameness, shoulder lameness can be detected by gait alone. the inability to "get the limb forward" being very noticeable when seen from the side as the animal trots. Trotting toward the examiner. the horse will exhibit a "sweeping outwards" with the affected limb. In time contraction of the heels follows shoulder lameness.

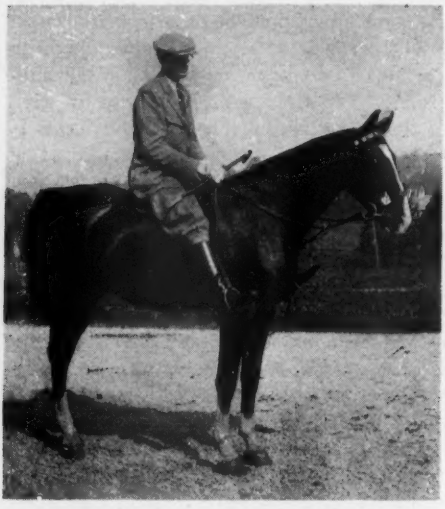
Fracture of the os pedis is occasionally observed but is of no significance in prerace examination as the animal is obviously too lame to run. One case of this type, in my experience, was rested for a year without any other treatment and then was able to win races the following year.

Tricks in shoeing or plating the horse's hoofs are of importance to veterinary examiners. Even the possibility of lameness caused by a corroding nail or too much sole pressure must not be overlooked. Before any treatment is decided upon, diagnosis should be as certain as possible. Makeshift "reliefs" can cause serious injuries in what might otherwise have been mild cases of lameness. It is my considered opinion that at least 90% of the cases where horses have been subjected to the barbarous "firing and blistering," treatment was unnecessary. They represent a sad commentary upon the progress of the veterinary profession.

Probably claudications due to soreness or strain of tendons and muscles cause the heaviest tax upon the ingenuity and judgment of a veterinarian. If he wishes to avoid injustice to the public, the owner or both, his examination must be painstakingly thorough. I have found it helpful to examine the horse while he is in a standing position, palpate his shoulders, limbs, joints and feet, then have the horse jogged away from me, then back, then turned to the left and to the right. Observation is made as to whether the lameness increases or decreases during the trotting. If it is slight and decreases, one is safe in allowing the animal to race, especially if he has not been campaigned, for this is the type of soreness which a horse will "run out of" and which will be forgotten in the heat of the battle. If the lameness increases, try to ascertain whether it is a swinging leg or a supporting leg lameness. Then locate, if possible, the spot from which the lameness originates. Make a note for future reference. Place the animal on the "veterinarian's list," which means that he must not be entered for at least a week, and then only after a report from the veterinarian to the stewards that he has recovered.

Taking note of the type of lameness is of

utmost importance to the examining veterinarian. The majority of owners and trainers and grooms are honest people who have a sincere love of horses. There are few who, aided by the pseudo-veterinarian (which Racing Commissions and racing authorities unfortunately do nothing to curb), use every means in their power to disguise or hide the pain. If one knows his case and the type of lameness from the first exam-



The late John F. Devine on his mount Shooting Star which won many blue ribbons

ination, one knows whether or not the lameness could possibly have been competely eradicated in such a short space of time.

There is essential knowledge that the race horse veterinarian must have: knowledge of racing people, grooms, trainers, owners, stewards and judges. He must have the courage of his convictions against sometimes impressive odds. If he has these two things, his professional ability will show itself at its full value. If he does not have them, his success will certainly be limited.

Racing Commissions can do—and have done—a great deal toward the improvement of the "sport of kings." The veterinarian's part in the elevation of racing has been, and always will be, a major part.

★ ★ ★ BUY WAR ★ ★ ★
STAMPS AND BONDS

The amount of tetanus toxin, per pound of body weight, required to kill a fowl is 350,000 times as great as for the horse. The tolerance of the dog, per pound of body weight is 600 times that of the horse. The sequel: tetanus in horses is common; in dogs rare; and in chickens non-existent.

The Horse Developed in America

It is a well known fact that the Spanish discoverers of America found no horses on this continent, and that the modern horse *Equus caballus* (Linn.) was subsequently introduced from the old world. It is, however, not so generally known that horses had formerly been abundant on this continent, and that long before, in Tertiary time, near relatives of the horse and probably his ancestors, existed in the far west in countless numbers, and in a marvelous variety of forms. Considering the remarkable development of the group through the entire Tertiary period, and its existence even later, it seems strange that none of the species should have survived, and that we are indebted for our present horse to the old world.—*O. C. Marsh* in *Fossil Horses in America.*

More Power From Horses

Horses will be used on many farms to furnish a part or all of the traction power necessary to meet production goals for food and fiber for some time. Farmers should not overlook the importance of getting the horses in top condition and health for the required work. Good feeding is essential but consideration should also be given to the methods of ridding the animals of internal parasites. These parasites lower the vitality and staying-power and also increase the susceptibility of infested horses to attacks of colic.

The best time to treat horses for parasites is during the winter months. Treatment may be given any time after the first frost but is often delayed until December or January. Carbon disulfide is effective in ridding horses of bots and roundworms; and phenothiazine is particularly effective in removing strongyles from the digestive tract.—Department of Animal Pathology and Hygiene, University of Illinois, Urbana.

The Clinical Diagnosis of Pregnancy in Cattle*

By J. W. BURGESS, M.R.C.V.S.
Royal (Dick) Veterinary College,
Edinburgh, Scotland

THERE is no need to stress the importance of the diagnosis of pregnancy in connection with the treatment of sterility. It is of primary importance, and everyone who intends taking up this work should be thoroughly conversant with the technic. Without this knowledge and experience grievous mistakes in diagnosis and treatment are bound to occur.

It is a fact that farmers and dairymen in most parts of the country have a firm belief that it is impossible to say with certainty whether or not an animal is in calf until the later stages of pregnancy. The blame for this can be laid only at the door of practitioners whose diagnosis in many cases is confined to ballottement or palpation of the fetus through the flank. On this account, many a good animal has gone to the slaughterhouse with a fetus inside her and, looking at the matter from the point of view of treatment for infertility, it is almost a certainty that numbers of animals have received an intra-uterine injection of iodine or some other substance, often unnecessarily, sometimes perhaps disastrously.

It was in view of the importance of having a practical knowledge of the handling of the genitalia of the cow per rectum that, a year ago, I commenced classes for fifth-year students of this College at a dairy farm near Edinburgh. In the limited time at my disposal it is, of course, impossible to turn out experts, but this class had at least the result of making the men enthusiastic and anxious to learn more, and I am certain many of them will continue to add to their knowledge whenever opportunity occurs.

One of the chief difficulties in instructing a large class in this work is the fact that only a small number can be taken on at a time. In many cases, after the animal has been examined once or twice, the rectum becomes atonic, ballooned and stretched, rendering palpation of the various parts difficult and in some cases impossible. These animals have to be left for a few minutes. Before, and for some time after commencing this class, I had fears that so much handling, especially by the more or less inexperienced, might have disastrous results. These fears proved to be quite unfounded. Animals settle down immediately afterwards and in some cases do not even stop feeding during the examination.

In this paper I propose to confine my remarks principally to the handling of the genitalia per rectum. This method is without doubt the most reliable means of diagnosis and I shall only refer briefly to the other signs which, though usually not reliable by themselves, are often of considerable value when taken together.

To begin with, the non-appearance of blood at the vulva after service (taken by some as indicating that conception has occurred) is unreliable. Blood, generally small in amount, is said to appear within three days of service if the animal has not conceived. Neither can cessation of estrus, the sign most relied on by breeders, be depended upon from our point of view. It must also be remembered that one may meet with cases where conception has occurred and the animal still comes into season, but as far as my experience goes these cases are uncommon. The presence of a uterine seal is generally given as a sign of pregnancy, but *per se* it cannot be relied upon, since in a number of cases it cannot be demonstrated in animals which are certainly pregnant. The palpation of a corpus luteum is of far more value, when considered with other signs, in animals which are examined at intervals. The palpation of the uterine arteries with the characteristic pulse in pregnancy is a valuable sign, and in most cases easily picked up. Auscultation of the fetal heart, as quoted in textbooks, can be definitely ruled out, as other and far more reliable signs are always available. The same may be said of palpation of the fetus per vaginam. Ballotte-

*Address to the Scottish Metropolitan Division N.V.M.A., meeting in Edinburgh. Reprinted from *Vet. Rec.*, 54:7, pp. 81-82.

ment or palpation through the flank is valuable in the later stages of pregnancy.

The remaining clinical method is palpation of the uterus and fetus per rectum, and, before I go further, it might be well to explain that no one need think that he can become proficient by merely reading up the subject. The most I can do in a paper such as this is to give hints which would help a beginner. It should be realized that there are quite appreciable differences in the genitalia of different animals—not only in non-pregnant but also in pregnant animals at the same stage of pregnancy. The ease or otherwise with which the uterus can be palpated also depends a great deal on the age, condition, size or breed. To describe each condition met with would not only lead to confusion, but would be impracticable. Practical experience is, therefore, absolutely necessary for any degree of accuracy in diagnosis, and fortunately, material to work on is within reach of everyone in country practice.

To those of you who have seen the Bayer film on the diagnosis of pregnancy and have not yet tried the technic, I might issue the warning that you may receive rather a shock on attempting the method. The film leads one to believe that it is all quite simple, due to the fact that the operator's hand, in many of the processes, is shown free in the abdominal cavity, without the rectum intervening to complicate matters. Nevertheless, the film is of undoubted value to the beginner.

To begin with, I strongly advise the thorough examination of the normal, healthy, non-pregnant uterus. During the examination little or no restraint is necessary. An assistant may stand alongside the animal and at the same time hold the tail, or the operator may hold up the tail with one hand and examine with the other; this, by the way, is an advantage in that it allows a closer approach to the anus. Only in exceptional cases is it necessary to have an animal held by the nose.

Another practical method for the examination of a patient for pregnancy and sterility. Every possible effort is made to maintain cleanliness

The operator, provided with a bucket of water and soap, lubricates his hand and arm and the anal region of the cow. Either hand may be used, but the left has been found most convenient. This hand is then inserted into the rectum. Straining, with contraction of the walls of the rectum, generally occurs at this stage. Allow the spasm to pass off before proceeding further and then clear the rectum of feces. A good method of emptying is to insert the tips of the fingers into the anus only and then

spread them out. The animal will then empty the rectum herself. Do not force the hand against any spasm, otherwise damage to the rectal mucosa is almost sure to occur, causing a harmless but undesirable hemorrhage.

Once the rectum is emptied, the cervix is now easily identified, lying on the floor of the pelvis in the middle line. Its shape, size and consistency should be noted, and there is never any difficulty in finding it. By moving the hand in a forward direction, one then encounters the bifurcation at the extremity. This is easily distinguished. By palpating with a finger on the dorsal surface one finds the groove between the two horns.

In the next step—palpation of the horns and ovaries—difficulty may be met. In heifers, for example, the whole of the genitalia, cervix, horns and ovaries can be covered by the palm of the hand with the hand about 9 to 10 inches within the rectum, *i.e.*, with the anus an inch or so above the level of the wrist. I mention these measurements because there is a tendency for beginners to put the hand too far forwards. In many animals which have had calves, however, the horns may sweep forwards out of reach, but it is always possible to palpate the ovaries, although the finding of the ovaries may give more trouble to beginners. The tendency, I find, is to search too high up in the pelvis. They are often misled in expecting to find both ovaries lying widely apart. More often they lie quite close together, and are low down in the pelvis, often almost level with or just above the level of the pelvic brim. In young animals there is never much difficulty. As a rule, I find that once a beginner has been able to find the ovaries in one or two cows, he can find them almost straight away later on. I do not permit students to use forceps to pull back the genitalia in cases where the horns are out of reach, but I find that the cervix and consequently the horns can be brought back into the pelvis, to a certain extent, by pushing a finger into the bifurcation and retracting per rectum. This brings the cervix back under the palm of the hand and it can be held there by pressure from above by the palm, thus leaving the fingers free to follow up the horns for comparison and examination. This comparison and examination I stress at this point, as it has an important bearing on the diagnosis of pregnancy in its earlier stages.

Other structures in the vicinity should be made out and examined. The fallopian tubes cannot be detected in a sexually healthy animal and, in any case, do not concern us here. The middle uterine artery

This cow evidently had been pregnant

can be picked up and its character should be noted. Other structures, *e.g.*, the rumen and bladder, should also be identified.

As many non-pregnant, sexually healthy animals as possible should be examined until the operator is thoroughly proficient at finding and palpating the various parts. The examination of a number of different animals will also demonstrate individual variation in position and size, thus safeguarding against mistakes in diagnosis later on.

Pregnancy diagnosis by palpation of the uterus can be carried out in most cows with a fair degree of certainty by the end of the second month, *i.e.*, eight weeks after service; in heifers, possibly a few weeks earlier. In some cows, however, unless one had other evidence and breeding history, there may be doubt at this stage; no actual bulging of the pregnant horn can be made out, but on comparison with the non-pregnant horn it has become more firm and turgid and a slight enlargement with a tendency to straighten out can generally be detected. In heifers there may be quite

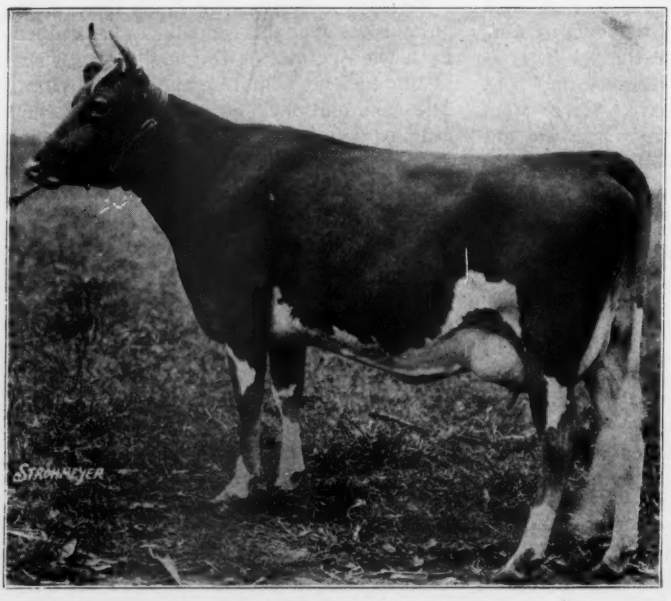
marked change as early as this, but I am of the opinion that it takes about two months before any appreciable change can be noted. It is therefore advisable to take all other available signs, together with breeding history, before making a diagnosis. Between the second and third month the change is more marked, though not perhaps so much as one would expect, but by the end of the third month there should be no doubt. The horn may be palpated along its length if retracted. Towards the end of the third month, bulging at the point in the horn to which the fetus is attached can be made out, the rest of the horn is still firm, and by now there is no difficulty in differentiating between pregnant and non-pregnant horns, on comparing the two. The enlargement of the uterine arteries also begins to be appreciable about this time.

From the third month on, growth of the fetus is rapid and by the fourth month a great change has taken place. Early in the fourth month the presence of fluids surrounding the fetus becomes evident, and later on the fetus itself can be palpated, not by grasping any part of it but by striking the uterus from the front and making the fetus rebound back on to the finger tips. By the end of this period the horn has enlarged enormously and is lying over the pelvic brim within the abdomen. This has the effect of pulling the cervix over the brim; the point where it crosses the brim can be easily determined. The pregnant horn is now lying slightly to the right of the middle line and the uterine arteries can be found with ease.

In the earlier part of the fourth to fifth month period, cotyledons can be palpated and later parts of the fetus itself can be made out. Still later on, the fetus may drop out of reach of the operator's hand. In these cases it is generally possible to palpate cotyledons and, from what I have said previously, there is plenty of other evidence.

By the end of the sixth month, the fetus is lying in the right flank, near or on the abdominal floor, and may be palpable through the flank. This palpation of the fetus through the flank should not be looked on as entirely dependable. Negative results from palpation do not mean that the animal is non-pregnant, and a rectal examination should always be made in doubtful cases.

I might point out that in some animals—particularly the smaller breeds—it is possible to palpate the fetus per rectum right on to parturition. In all animals the fetus can be palpated easily per rectum just before parturition, and since it is at this stage that palpation per vaginam is sometimes suggested, I would certainly recommend rectal palpation in preference.



Finally, I should also like to express the opinion that to get the best possible results in the treatment of infertility, visits to the farm would have to be at far more frequent intervals than is suggested, *viz.*, every three months, as it is in the early diagnosis and treatment of infertility that we can help our clients most.

* * * *

Crystal Violet Vaccine in the Prevention of Hog Cholera

By E. F. SANDERS
and A. H. QUIN*
Kansas City, Mo.

ACTIVE immunity, produced in cholera-susceptible swine by simultaneous inoculation of anti-hog cholera serum and hog cholera virus, is of extremely high quality. Swine so immunized may be hyperimmunized by intravenous injection of 5cc of virulent hog cholera virus for each pound of body weight, the equivalent of many million lethal doses for unvaccinated, cholera-susceptible animals.

Field application of such dependable immunity has made possible the growth and development of our American swine industry. However, the injection of fully virulent virus and potent serum is attended by certain inherent risks, not all of which are avoidable. Herds affected by coincidental bacterial infections, by gross parasitisms or by severe nutritional disturbances may suffer over-reactions due to injected virus even though the conjoined dose of serum is ample under routine field conditions.

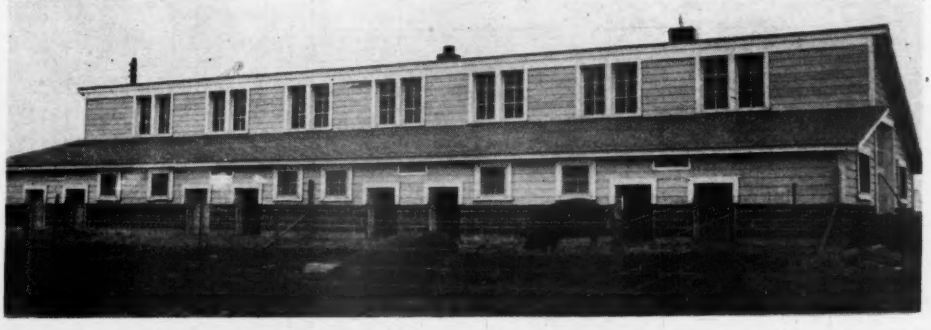
It is not always possible for a veterinarian to recognize masked infections in a herd just prior to serum-virus usage. Neither does the practitioner have control over adverse climatic conditions, feeding errors and other factors which may influence the general health level of swine during the reaction period. It is also well known that pigs quite regularly develop a marked leucopenia subsequent to serum-virus inoculation. This factor may permit enhancement of virulence by *Salmonella choleraesuis, Pasteurella suisepticus,* and other pathogens.

From the standpoint of veterinary sanitary regulations and disease control, the establishment of new foci of hog cholera on premises where herds suffer over-reaction losses is a most serious problem, especially in areas where the disease is a sporadic menace rather than a perennial hazard.

Need for a Nonviable Immunizing Agent

The aforementioned disadvantages of serum-virus hog cholera prophylaxis have spurred veterinary scientists in this and other countries to search for a nonviable, avirulent cholera preventive. In America, this research was pioneered by the late Dr. Marion Dorset, chief of the Biochemic Division of the Bureau of Animal Industry of the U. S. Department of Agriculture, and by Dr. W. H. Boynton, of the University of California. Following Doctor Dorset's death in 1935, continued and extensive laboratory and field studies of crystal violet hog cholera vaccine have been carried on by Drs. C. N. McBryde and C. G. Cole at the B.A.I. Field Station at Ames, Iowa. Since Dr.

*Director, Biological Farm, and Head, Department of Clinical Veterinary Medicine, Jensen-Salsbery Laboratories, Inc., Kansas City, Mo.



New half-monitor farrowing house at the U. S. Department of Agriculture regional research farm, Ames, Ia.

McBryde's retirement, Doctor Cole has been in charge of the project. Over the past 10-year period many thousand head of swine have been immunized with crystal violet vaccine under field conditions. Hundreds of these pigs have been checked by being given an inoculation of virus at the time they were ready for market. The immunizing efficiency, by all reasonable angles of analysis, has proved to be quite satisfactory, as well as safe.

The questions relative to a nonviable hog cholera vaccine uppermost in the minds of veterinarians are: (1) What proof is there that a crystal violet vaccine will protect pigs against exposure to, or inoculation with, hog cholera virus? And (2) What are the indications and contra-indications for such a vaccine in practice?

A Potency and Safety Test

In answer to the first question, an experiment recently conducted by the writers may be of interest. Three sample lots of crystal violet hog cholera vaccine, blood origin, were prepared in strict accordance with the production specifications outlined by the Federal Bureau of Animal Industry.

Thirty-six susceptible pigs, weighing from 50 to 80 pounds each, were obtained from a farm and brought to the laboratory in thoroughly disinfected trucks. They were divided into three groups of 12 each (one group for each lot of vaccine to be tested), isolated from other swine and held under observation for a period of two weeks in order to prove freedom from any incubating ailment.

At the end of the holding period, each lot of the vaccine was tested by the use of 12 pigs as follows:

Seven pigs were given 5cc of the vaccine, two received 3cc each and the remaining three pigs were left in the pen with the vaccine-treated animals to serve as controls at time of exposure to virulent hog cholera virus.

Three weeks following vaccination, each vaccinated pig, as well as each control pig, was inoculated with 2cc of virulent hog cholera virus.

The accompanying protocols may be summarized as follows:

Eight of the nine control pigs manifested the climax of acute cholera and were killed on the seventh day following virus inoculation.

One control remained refractory throughout the test period.

All 21 pigs receiving 5cc of the vaccine remained well 14 days following virus inoculation.

It is especially significant to note that the six pigs receiving only 3cc of vaccine each withstood a challenge dose of 2cc of virulent hog cholera virus. This gives con-

Potency Test on Crystal Violet Vaccine—Lot No. 1

Pig No.	Lbs. Wt.	Date Vaccinated	Dose Vaccine	Method	Date Exposed	Dose Virus	Observations After Virus Injection — DAYS — 1	2	3	4	5	6	7	8	9	10	11	12	13	14
26	55	4/2/43	5 cc.	Subc.	4/24/43	2 cc.	SUNDAY	√	√	√	√	√	√	SUNDAY	√	√	√	√	√	√
27	60	"	"	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
30	60	"	"	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
31	65	"	"	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
32	55	"	"	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
33	60	"	"	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
34	80	"	"	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
37	55	"	3 cc.	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
38	50	"	"	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
29	50	—	Control		"	"		√	√	√	S	S	S	K						
35	70	—	Control		"	"		√	√	√	S	S	S	K						
36	70	—	Control		"	"		√	√	√	S	S	S	K						

√—Well. S—Sick. K—Destroyed.

Potency Test on Crystal Violet Vaccine—Lot No. 2

Pig No.	Lbs. Wt.	Date Vaccinated	Dose Vaccine	Method	Date Exposed	Dose Virus	Observations After Virus Injection — DAYS — 1	2	3	4	5	6	7	8	9	10	11	12	13	14
39	55	4/2/43	5 cc.	Subc.	4/24/43	2 cc.		√	√	√	√	√	√		√	√	√	√	√	√
41	65	"	"	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
43	65	"	"	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
45	60	"	"	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
47	60	"	"	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
48	60	"	"	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
50	75	"	"	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
46	60	"	3 cc.	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
49	75	"	"	"	"	"		√	√	√	√	√	√		√	√	√	√	√	√
40	70	—	Control		"	"		√	√	√	S	S	S	K						
42	42	—	Control		"	"		√	√	S	S	S	S	K						
44	75	—	Control		"	"		√	√	S	S	S	S	K						

SUNDAY

√—Well. S—Sick. K—Destroyed.

crete evidence of the antigenicity of the vaccine.

Limitations

It is generally agreed that about 21 days must elapse, following crystal violet vaccine injection, before swine reach the zenith of their immune response. Under widespread usage, it may become apparent that sufficient protection has been built up by the 14th day to withstand ordinary field exposure. This time interval must, however, be kept in mind for during this interim every effort must be made to prevent accidental exposure of the vaccinated pigs to hog cholera virus. Furthermore, particular precaution must be practiced by the veterinarian to insure that no clothing, syringes, needles nor other equipment are contaminated by virus at the time pigs are treated with vaccine. No garbage should be fed to treated pigs for three weeks following vaccination.

All available research indicates that a single dose of crystal violet vaccine will

Potency Test on Crystal Violet Vaccine—Lot No. 3

Pig No.	Lbs. Wt.	Date Vaccinated	Dose Vaccine	Method	Date Exposed	Dose Virus	Observations After Virus Injection — DAYS — 1	2	3	4	5	6	7	8	9	10	11	12	13	14
2	75	7/7/43	5 cc.	Subc.	7/28/43	2 cc.	√	√	√		√	√	√	√	√	√		√	√	√
5	75	"	"	"	"	"	√	√	√		√	√	√	√	√	√		√	√	√
7	60	"	"	"	"	"	√	√	√		√	√	√	√	√	√		√	√	√
8	70	"	"	"	"	"	√	√	√		√	√	√	√	√	√		√	√	√
10	55	"	"	"	"	"	√	√	√		√	√	√	√	√	√		√	√	√
11	70	"	"	"	"	"	√	√	√		√	√	√	√	√	√		√	√	√
12	75	"	"	"	"	"	√	√	√		√	√	√	√	√	√		√	√	√
3	65	"	3 cc.	"	"	"	√	√	√		√	√	√	√	√	√		√	√	√
6	75	"	"	"	"	"	√	√	√		√	√	√	√	√	√		√	√	√
1	80	—	Control		"	"	√	√	√		S	S	S	K						
4	70	—	Control		"	"	√	√	√		√	√	√	√	√	√		√	√	√
13	60	—	Control		"	"	√	√	√		S	S	S	K						

SUNDAY

√—Well. S—Sick. K—Destroyed.

protect pigs up to time of marketing (usually eight months). Any animals held over for breeding stock or other purposes should be revaccinated within eight months after the original or first vaccination.

Crystal violet is contraindicated for exposed herds, for feeder pigs without definite history, and for nonimmune pigs on garbage feeding premises.

Advantages

Widespread, year-by-year immunization of swine by the crystal violet vaccine method—if coupled with suitable livestock regulatory measures—should bring nearer the ultimate goal of hog cholera eradication. By this method the recurring flares of "virus out of bounds" should be reduced to a minimum.

The fact that the dose of hog cholera vaccine is the same (5cc) for swine of all ages and weights is also a distinct advantage.

From the veterinarian's standpoint, crystal violet vaccine is a totally sterile product. Thus any risk of transmitting viruses or pathogenic bacteria with the vaccine is eliminated.

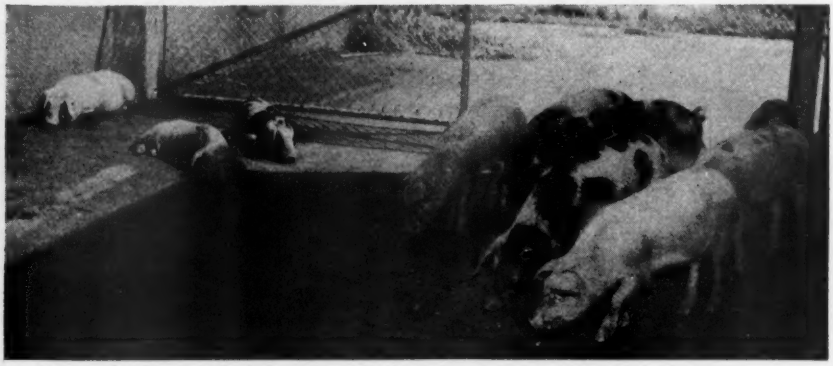
Because it is wholly avirulent, hog cholera vaccine can be used to confer protection on herds that are unsuited for serum-virus prophylaxis. This insures against total herd loss by hog cholera while other infections or herd troubles are brought under treatment. It also eliminates cholera as a complicating factor, thus making diagnosis of other diseases, notably swine erysipelas, less difficult.

From the owner angle, the avoidance of reaction setbacks and the ability to keep the herd on a full feeding schedule after crystal violet vaccination are most desirable advantages for, by this means, pigs may be finished in less time and at lower costs.

Conclusions

These experimental lots of crystal violet hog cholera vaccine, blood origin, prepared according to the production methods outlined by the U. S. Bureau of Animal Industry, protected pigs in a dosage of 3cc and 5cc against a challenge dose of 2cc of virulent virus given three weeks later. Eight of nine control pigs given 2cc of virus alone, and kept in contact with the vaccinated pigs, sickened or were killed following development of typical hog cholera. One control pig remained refractory throughout the test period.

Crystal violet vaccine should bring nearer to fruition the ultimate eradication of hog-cholera for, if the critical test of widespread field usage confirms present observations, the "flares" due to virus over-reactions may be eliminated or reduced to a minimum through careful use of this non-viable immunizing agent.



OFFICIAL TEST ON CRYSTAL VIOLET VACCINE

Five of the seven pigs at right received 5cc vaccine; two received 3cc. Three weeks after vaccination these seven pigs, together with three unvaccinated control pigs at left, were given 2cc virulent hog cholera virus. The three control pigs are prostrated with typical hog cholera, while the seven vaccinated pigs remained well. All 10 head were in intimate contact in the same pen during the five weeks of the test period

"Housedog" Disease

By LEON F. WHITNEY
Orange, Conn.

THE term "distemper" was formerly a catch-all for many debilitating febrile diseases of dogs. That was before the isolation of the filterable virus by Carré. Since then, we are coming to see that distemper of pre-Carré days was actually a group of diseases having somewhat similar symptoms. Complete unscrambling of these diseases is not yet accomplished. But a sound understanding of them and their differences is essential to proper diagnosis, treatment and prognosis.

Much has been written on the subject of true distemper—the disease caused by the virus of Carré. But the diseases formerly included with distemper have received less consideration. Ferry, Slingman and Frohring have made notable contributions to the subject, which are well known. Some time ago this writer described one of the "distemper complex" calling it canine influenza.[1] This paper deals with still another disease of the distemper group, for which, until more is known about it and the specific cause determined, the name "housedog disease" is suggested, since, so far as observations go, it is preeminently a disease of dogs living in intimate contact with persons in the household. Clinically it is a pharyngo-laryngo-trachitis.

It is not introduced as a new disease. How long it has existed unrecognized is problematical. In this section it goes under many names—distemper, nervous distemper, nervous form of distemper, strep sore throat, septic sore throat, cold, coryza and influenza. Much newspaper publicity was given here in Connecticut recently to a disease described as encephalitis. It was said that a new dog disease was sweeping sections of the state. The prevalence of housedog disease and the high incidence of encephalitis as a complication led to this belief that a new disease was decimating our canine population.

The writer has practiced in this district only three years. Housedog disease has been very prevalent all of that time. Possibly it has long been confused with true distemper, and since it is usually a mild affection, prone to spontaneous recovery, it may account for the many "cures" of "distemper" by small doses of serum, or by the use of sulfanilamide, vitamin administration and other remedies which do not cure the real Carré distemper when put to the test with adequate controls.

About 800 cases of housedog disease have been brought to the writer's clinic in the past two years. In this time, just eight cases of genuine Carré's distemper have been presented. Perhaps it should be mentioned that my experience with Carré's disease is not limited to these eight cases. During the years when engaged in canine research over 500 cases of true Carré distemper occurred in my own kennels and a greater number were studied in other kennels. During the past two years calls have come from private kennels where there were epizootics of Carré distemper, but even these outbreaks have been few compared with the number which occurred in the last decade. Laidlaw and Dunkin[2] pointed out very forcibly the necessity of using what they called "compound bred" dogs in distemper studies, to avoid confusion with other diseases. Anyone working with diseases of the distemper complex has it brought home to him every day, how right they were, and how almost impossible it is to make an accurate diagnosis from one short period spent with a dog in a veterinary hospital or from a visit in a home. Even observation of a dog for several days in a hospital may not be sufficient time for a correct diagnosis.

Because of this difficulty of diagnosis, it is possible that some of the dogs included in this study may have been suffering from some other disease. Indeed, it is probable that such is the case but the number is small, as there was a careful follow-up wherever possible. This study covers 309 of the more than 800 cases observed; these

[1] Whitney, Leon F., 1941. Canine influenza. *Vet. Med.*, 36:4, pp. 216-219.

[2] Laidlaw, P. P., and G. W. Dunkin, 1928. *Jnl. Com. Path. & Ther.*, 41.

being the cases where the entire history is known and a complete follow-up was carried out. Doubtful cases are not included, nor are those on which insufficient data

TABLE I—INCIDENCE OF HOUSEDOG DISEASE AND DOGS WITH SUBSEQUENT ENCEPHALITIS BY AGE GROUPS.

	Housedog Disease	Encephalitis	Percentage
1-6 months	74	27	36
7-11 months	67	15	22
1 year or more	168	16	9
Total	309		

were recorded as, for instance, omission of the temperature, of the dog's age or the onset of the disease, etc.

It is important to understand that this disease is not encephalitis—or fox encephalitis as that disease is known where it is the primary infection. This type of encephalitis is one of the sequels of housedog disease.

During the past two years housedog disease appears to have been the most important disease of the canine population of Connecticut. At the Whitney Clinic it stands second only to skin diseases, as a reason for presenting dogs for treatment. Several friends of the clinic at Yale Medical School became interested in the disease and as a result of their valued cooperation, a better syndome is presented. The section of this paper dealing with the brain pathology was written by Dr. H. M. Zimmerman of the Yale school.

Description of Housedog Disease

This disease, when uncomplicated, is a mild one. A large proportion of the cases brought to our attention were introduced either in a telephone conversation, or an office call, with the words, "Doctor, my dog has a bone stuck in his throat." The outset is recognized by the owner by shivering and a cough or retching, at which time the animal appears to vomit white froth. In very severe attacks the froth may be tinged with blood. Retching may continue until some of the contents of the stomach are brought up, or attacks may be so mild that the animal simply coughs up or appears to vomit a small amount of white phlegm which he leaves here and there about the house. Some dogs swallow the froth, acting much like a puppy that is heavily infested with hookworm or roundworm larvae.

Looseness of the bowel movement is generally associated with the coughing symptom. In a few instances some convulsions occur. Stethoscopic examination of the lungs sometimes reveals a whistling sound, indicative of inflammation of the bronchi. These symptoms may disappear after four to six days, or the coughing may persist for weeks, depending upon whether the pharynx and larynx are left inflamed, and kept in that condition by the irritation of the coughing. About the seventh day pneumonia sometimes ensues, but more often recovery is uneventful.

On careful inspection the throat is seen to be inflamed; the soft palate may be mottled, or crimson, normal in texture or thickened; the tonsils are usually inflamed, sometimes standing out like a coxcomb, and of about the same color and they often touch across the back of the throat. Sometimes, but not always, the gums are inflamed when the involvement is intense. Froth may usually be found filling the tonsillar crypts or clinging to and around the tonsils, if they are enlarged and protruding from the clefts.

Palpation discloses considerable enlargement of the parotid gland and a distinct firmness, which is not present in the uninfected gland. Frequently several enlarged lymph glands may be felt forming a chain with the parotid. In a few cases, in larger breeds, these three glands on each side were enlarged almost to the size of hen eggs. Gross post-mortem examination of the whole upper respiratory tract shows involvement of the pharynx, larynx and trachea—the mucosa of all being thickened.

The temperatures vary more or less with age. Young dogs have much higher temperatures than older dogs which commonly show quite uniform temperatures, varying not more than a few tenths of a degree from 102° F.

If pneumonia does not follow the throat phase of the disease, the dog may appear to be entirely well in a few days. However, if temperatures are taken it may be learned that recovery is not complete. The temperature may be from a fraction to several degrees above normal. In such cases, from a week to a month later, the owner reports

that the dog has had a fit, or perhaps a twitch, or some other indication of encephalitis. If it be a convulsion, there may be but one that day. The next day there may be two or three or four convulsions. The next day the fits may be frequent and by the following day almost perpetual, and death usually ensues. In other cases the fits may continue for two weeks in a mild form, gradually weakening the victim. In at least a dozen of the 309 cases included in this report the fits began, lasted two or three days and ceased, only to recur after a few days' respite.

Twitches may start by the dog holding up a paw as though it pains. This may become more and more intense until the periodic jerking characteristic of chorea starts. Then again the chorea may start suddenly. The twitch may be only the winking of an eye, the jerking of a lip, or may be so violent as to affect both front legs and cause rhythmic contraction of them, letting the dog up and down, and jerking throughout even sleeping periods.

A rather odd characteristic of dogs afflicted with encephalitis is the good appetite which most of them have up to the approach of death.

For most dogs the disease is a simple one, not much more severe than is a cold in a person. In fact, it is not even of sufficient consequence to prevent bitches from coming in heat normally and continuing their season and mating successfully. Nor does it appear to cause resorption of fetuses as is often the case with distemper.[3]

Forms of Encephalitis Encountered

The following are the kinds of encephalitis so far encountered, all of which followed the primary disease described above:

1. *Convulsive Form.*—The animal may stand or sit in one place and suffer the common type of convulsion, becoming rigid, champing the jaws, or holding them open as if having difficulty in closing them. Saliva may become frothy and drip from the mouth. The dog usually falls on his side, and ends his convulsion in that position; finally becoming limp and, when arising, walking in a dazed manner. This condition takes some time to wear off. He often appears blind and deaf for many minutes following a convulsion.

Some dogs manifest typical running barking fits, in addition to the symptoms described above. Several dogs in which the convulsive type of encephalitis developed, simply sat in their cages, toward the end of the disease, acted wild and drooled saliva; reproducing exactly the textbook picture of rabies. They can be mistaken easily for rabid dogs.

2. *Chorea.*—The regular twitching of groups of muscles or of single muscles, often follows housedog disease. In some cases these symptoms developed almost suddenly, while in others they came on more slowly, as though there had first been an ache in the part. But the dog is left with the jerk that is typical of chorea.

Sometimes a dog suffering the convulsive type of encephalitis can be mistaken for a rabid dog as the animal pictured above. Toward the end of the disease the dog will act wild and drool saliva—a textbook picture of rabies!

3. *Tics.*—These are probably the same thing as chorea but in smaller groups of muscles. Sometimes only a lip is affected, sometimes the twitching is limited to one or more orbital muscles so as to cause the dog to wink constantly; sometimes one whole side of the face is involved.

4. *Spastic Paralysis.*—Only two such cases have been seen in our clinic. One was a great Dane which lived; the other a mongrel which died. In both cases all skeletal

[3] Whitney, Leon F., 1936. Resistance to distemper in the pregnant bitch. *Vet. Med.*, 31:6, pp. 254-258.

muscles of the dogs became rigid. To distinguish this from a brain tumor, nembutal was administered and the animals became limp.

5. *Lethargic Form.*—Six dogs with this type of the disease have been encountered. Three of these died and three survived. So far as their digestion and elimination were concerned, they might still be alive had there been any point in keeping them. These animals went into a profound sleep from which it was difficult to arouse them, even for feeding. Once aroused, they ate well. They lived two months and grew physically stronger. Their minds were blanks. One even had to be held over his food so he could eat and then wolfed it. His eliminations had to be attended to several times a day and he kept himself constantly soiled, and could not be trained in any way. The other survivors were not as badly affected as the first and could eat without help, but were very unclean.

6. *Vertigo.*—One German shepherd would lose his balance and suddenly drop on his side and then perhaps make a turn—sometimes several turns. As the encephalitis grew more severe, at one time, he would make as many as four corkscrew turns in his cage. A little excitement would bring on an attack. After three days of this he developed convulsions associated with the twisting and died during the night. Several other dogs twisted their heads only and if they were picked up would appear to be trying to make corkscrew turns, the head leading the body. When placed in their cages, they would not roll over, but preferred to lie on only one side.

7. *Ophthalmia.*—Those animals which became blind as a result of housedog disease exhibited various symptoms. Three had such dilated pupils that it is assumed the retinas were destroyed. Three had normal pupils but were blind. No post-mortem examination was made on any of these. One dog appeared to have a chorea-like twitch in the oblique muscles of both eyes. His eyes jerked sidewise with clocktick regularity, annoying the owner so much that she had the dog destroyed.

8. *Disorientation.*—A common type of this ailment is best described by the expression, "startled attitude." The dog stands looking as though he were startled. This same attitude and expression is seen preceding the convulsive type, but in the type here described, it does not go beyond the startled appearance. The dog usually fails to obey commands and does not recognize his owners. This type sometimes recovers after several days.

Some of these cases of encephalitis possibly suffer from an intense cephalgia. In such, if the dog is treated as though he had a splitting headache, his lot may be made easier. Loud noises, excitement, bright light and so forth have a tendency to increase the pain and should be avoided.

Incubation Period

The incubation period appears to be short in housedog disease. It varies somewhat, but is usually about two or three days. Apparently the virus is taken into the nose, and establishes itself in the mucosa of the throat. It appears to spread rapidly, causing an irritation which in turn causes the dog to exhibit the symptoms of a sore throat. At the onset, the throat is not observed to be inflamed, the tonsils usually do not show but are hidden in their clefts. Then, as the disease progresses, the pharynx appears to be more or less inflamed, varying with the severity of the attack.

Determination of the exact incubation period of housedog disease has been difficult since it does not spread with the certainty of Carré distemper. Often only one of several dogs in a home becomes infected. Repeatedly we have observed one of a pair of puppies contracting the disease and the other not affected for two or three days.

Susceptibility

Among puppies in homes, we have not seen any case where one has had housedog disease and the others have not contracted it, if they were left together. This cannot be said for grown dogs for, as stated, there are many instances where one dog has had the disease and another living constantly with it, has not contracted it.

As has been said, the disease is confined almost wholly to housedogs. It has been most interesting to see this fact so clearly demonstrated. So far not a single dog that

had been kept out of doors the year around is known to have been affected. A large outdoor kennel is maintained adjacent to our main hospital. Dogs kept at the hospital are segregated, when they have housedog disease. At times these dogs have been of them to a series of shows. Upon returning home all of these dogs developed symptoms of the disease and some died from encephalitis that subsequently developed, but the disease did not spread among the other dogs of the outdoor kennels.

Dogs kept in out-door kennels do not contract housedog disease even when dogs having the disease are allowed to run with them

walked deliberately around this kennel where our own dogs are kept. No attempt has been made to prevent transfer of the disease, and yet not a case has occurred among the kennel dogs. Imagine what would have happened had Carré distemper been treated as lightly among a kennel of susceptible dogs!

In four instances some of these kennel dogs were brought into the hospital ward in which dogs with housedog disease were being treated. All of them contracted the disease within a few days after which they were put out into the kennels again and allowed to run with their kennel mates—these did not contract the disease. Recently six cocker spaniels from the kennel group were spayed and given away. All went into homes. Up to the present writing five of the six have been returned with the disease, for treatment.

Animals that attend an indoor dog show, often develop the disease. A friend who runs a large kennel of sled dogs took six

Source of the Infection

When one attempts to ascertain the *modus operandi* of the spread of the disease, one is struck by the almost certainty that it is contracted both from persons and from other dogs. To put it another way, a house dog may contract the disease from persons suffering from certain types of colds or from affected dogs. On one day five different dogs were brought to the clinic, not one of which had been near any other dog and in every case one or more members of the family were suffering from colds at the time. Naturally a great many people have colds at any given time, especially in February when the incidence of housedog disease is greatest. A Gallup poll revealed that last year 25,000,000 persons in the United States had colds during the month of February. So it would be difficult to find a home without a cold. Nevertheless, one is struck by the fact that none of the five dogs mentioned contracted the disease until colds became prevalent in the household of which they were a part.

Pathological Changes in the Brain*

Gross.—Examination of the brain and meninges gives no conclusive proof of the existence of inflammation. The brain is usually swollen, soft and boggy; its meninges somewhat turbid but containing no frank collection of purulent material. The brains may appear angry-red in color from capillary congestion, but hemorrhages are rarely present. In the majority of cases when the brain is sectioned there are seen minute, pin-head, red foci of discoloration which are numerous in the white matter of the cerebellum, scant in number in the cortical and central grey matter and present in large numbers in the pons, medulla and upper portion of spinal cord. With increasing age of the cerebral affection, the red color of the lesions becomes altered and is gradually changed to brown.

Microscopic.—Dogs that succumb acutely to the virulent form of the encephalitis have numerous tiny hemorrhages surrounding the capillaries and venules. It is these hemorrhages which produce the red spots seen with the naked eye. The cerebral meninges may contain small collections of lymphocytes and similar cells may form collars around blood vessels in the cortex. But usually this evidence of inflammation is not conspicuous.

Animals which survive the onset of the brain injury for weeks, reveal a somewhat different picture on microscopic examination of their brains. There is usually extensive destruction of the ganglion cells in many of the cerebral gyri. These cells have either disappeared or are in different stages of disappearance. Some cells show "ischemic," liquefactive changes whereas others are shrunken and hyperchromatic. This loss of neurones is partially marked by a proliferation of microglial elements and by the cortical infiltration of lymphocytes. All the vessels in such areas of injury in the brain contain large numbers of small round inflammatory cells in their sheaths. The overlying meninges are richly infiltrated with lymphocytes. The general microscopic appearance of the cerebral cortex is similar to that of general paresis in man. None of the ganglion cells, injured or otherwise, contain any inclusion bodies. Perhaps the most widespread cellular, inflammatory reaction occurs in the brain stem, with especial involvement of the nuclei of the medulla.

The brain of an occasional animal discloses a variation of the microscopic picture of the lesions as described above. The variation consists of predominant involvement of the subcortical white matter of the cerebrum, the pyramidal fibers in the pons and the central medullary substance of the cerebellum. There is myelin sheath destruction with the formation of fat-granule cells. A mild glial proliferative change ensues with the formation of ectocystic nodules. In the cerebellar folia the central medullary substance is destroyed, the granular cell layer is slightly injured, with disappearance of some of the small neurones, and the Purkinje cell layer is spared. A few focal nodules of glia are scattered in the molecular layer of the cerebellar cortex.

In the spinal cord both the fiber tracts and the central gray matter are usually involved. The white matter suffers medullary sheath destruction, whereas the gray matter is involved in a cellular inflammatory process. The blood vessels have swollen endothelial cells and lymphocytic collections in the perivascular spaces. Microglia, leucocytes and lymphocytes replace much of the central gray. Miliary glial nodules replace some of the anterior horn cells of the spinal cord, but true neuronophagia is not present. Again there are no inclusion bodies, either nuclear or cytoplasmic. The spinal cord lesions are reminiscent of acute anterior poliomyelitis.

The central nervous system lesions of these dogs can be best described as a nonsuppurative encephalomyelitis with considerable demyelination. They are similar to certain of the encephalitis lesions of known virus etiology, but in themselves are not pathognomonic of any known virus. Thus their etiology must await solution by transmission experiments.

CHART I.—MONTHLY INCIDENCE OF HOUSEDOG DISEASE OVER A PERIOD OF TWO YEARS BEGINNING MAR. 1, 1941

Jan	Feb	Mar	Apr	May	June	July	Aug	Sept	Oct	Nov	Dec
44	60	28	20	15	12	15	12	15	19	25	44

Onset of Encephalitis Variable as to Time

The number of days between the onset of the pharyngeal symptoms and the initial encephalic symptom vary widely. This may appear, from the records, to be greater than the variation really is. The record is based on the word of the owner and he may not always have recognized the disease on the first day, often not until the dog appeared quite sick. Attempts were always made to ascertain the first day, but that fact could not always be accurately

*By H. M. Zimmerman, M.D., Yale Medical School.

determined. This doubtless accounts in part for the variation mentioned.

The shortest time elapsing between the onset of pharyngeal symptoms and the appearance of encephalitis was seven days, and the longest 37 days. No significant curve could be drawn from the 58 cases but it is interesting to note that the time elapsing did not fall in a peak but was scattered as follows:

Days	7	8	9	10	11	12	13	14
Number	4	1	0	2	3	4	5	2
Days	15	16	17	18	19	20	21	22
Number	1	2	2	3	3	2	3	1
Days	23	24	25	26	27	28	29	30
Number	6	0	1	0	2	0	0	0
Days	31	32	33	34	35	36	37	
Number	3	1	1	1	0	2	1	

It was noteworthy that the temperature shown by the dog during his throat phase of the disease did not form a basis for prognosis. It might be expected that the higher the initial temperature, the greater the likelihood of the development of encephalitis, but such was not the case. The temperatures were studied for age groups also. There was a somewhat higher temperature in the dogs that developed encephalitis in each of the three groups but several dogs showed temperatures over 105° F. and still did not develop the disease; whereas some with relatively low temperatures did develop encephalitis. There was no greater difference between the average of the groups than 0.7° F., and that was in the 1 to 6 months of age group.

Differentiation

It is necessary to differentiate housedog disease from Carré distemper and canine influenza. In Carré distemper there is the five-day incubation period, the long course (when secondary diseases are included), the characteristic temperature curve, the copious discharge from the eyes and nose, the photophobia and other symptoms well known to all veterinarians.

Housedog disease, by contrast, runs a short course; produces little or no mucous discharge from eyes and nose; is accompanied by relative slight elevation of the temperature (seldom far from 102° F. in mature animals) and photophobia is lacking. Phlegm or froth is coughed or gagged in contrast to the dry cough of Carré distemper. The disease seems to be localized in the throat, and there is very much less prostration than in Carré distemper.

Solid immunity to the Carré's disease affords no protection against housedog disease. This fact is of great concern to veterinarians because many dogs, which have been immunized against Carré distemper, later contract housedog disease and the owners are convinced that their dogs are affected with a light form of distemper and that the anti-distemper vaccination given was inadequate.

A further and certain differentiation is the fact that no inclusion bodies are found in the epithelial cells of dogs with housedog disease whereas Carré distemper is characterized by such intracellular inclusions. The encephalitis likewise is not accompanied by endothelial inclusions as is fox encephalitis.[4]

In contrast to canine influenza, we have these differences: In influenza there are no severe throat symptoms, but general prostration, with a high temperature that continues for four or five days after it has reached its peak, and then drops suddenly to just above normal. A dog having had distemper is immune to influenza, whereas such is not the case with dogs affected with housedog disease. Encephalitis did not follow influenza in any of the cases observed, in the Whitney Kennels, but it is a common sequel to housedog disease.

Etiology

But little is known of the etiological agent. Presumably it is a virus, since repeated throat washings failed to demonstrate pathogenic bacteria but this is by no means proved. Definitely it is not a streptococcus organism, despite the fact that the disease is frequently diagnosed hereabout as "strep sore throat." Even tonsils of sick dogs which were triturated, emulsified, and plated by a competent bacteriologist revealed no pathogens.

A large number of mice were injected, sprayed nasally with blood, serum or throat washings and all failed to contract the disease. Dr. Paul Wenner of the Yale Med-

[4] Green, R. G., 1936. Fox encephalitis. *Vet. Med.*, 31:12, pp. 527-531.

ical School attempted to pass it to guinea pigs, mice and rabbits and none contracted it.

Puppies injected hypodermically with filtrate and emulsion made from tonsils of a sick dog, failed to contract the disease, but puppies left indoors in contact with the sick dog did contract it. So did older dogs—age was no barrier. This of course is evidence against the virus theory as to the causative agent, but the characteristics of viruses are so imperfectly understood that failure to infect in a single trial can be given little weight.

Another reason for assuming the disease to be a virus is because the sulfa drugs have proved so utterly worthless in the treatment of it. Sulfanilamide was studied[5] by Dr. Barbara Dickerson and the author against Carré distemper in 1938 and found to be almost worthless. And this is the case with all known viruses against which sulfa drugs have been tried.

In passing it might be noted how easily one could make out an excellent case treatment of housedog disease with the sulfa drugs. Let us say one of these drugs is prescribed and given for four days; the dog recovers. This happens in 100 cases. So the drug gets credit. But where an equal number of untreated controls are kept, they too recover without the drug. It is probable that reported cures of distemper by the use of sulfa drugs were "cures" of housedog disease, and the same may be true of many "cures" of Carré distemper with serum. Here is a confusing disease that can lead to erroneous conclusions unless differentiated.

[5] Dickerson, B. C., and L. F. Whitney, 1938. *Jnl. Exp. Biol. & Med.*, 38, p. 263.

Six sled dogs were taken to a series of shows. Upon returning home all developed symptoms of housedog disease and some died of encephalitis which followed. However, the disease did not spread to the other dogs in the outdoor kennels.

Breed Susceptibility

There seems to be considerable breed difference both as to susceptibility to the primary disease and to the subsequent encephalitis. Individuals of the wire-haired breeds are among the most numerous in the canine population and yet we have observed very few with housedog disease, and only two with encephalitis.

Cocker spaniels are the most popular house dogs and, as might be expected, they lead all other breeds in the number of cases of housedog disease. Mongrels, despite their many virtues, are more often kept by people who are not so careful of their dogs as are the owners of purebred dogs. But a considerable number of mongrels have been presented for diagnosis.

The English setters (kept in large numbers), the fox hounds, beagles and other hounds, as a general thing, are kept out of doors but, despite their large numbers among the dog population of New England, few have been presented.

Following is the breed assortment by numbers of the 309 dogs included in this study.

Cocker spaniel	70	Irish terrier	4
Mongrel	40	Poodle	3
Irish setter	17	Dalmatian	3
Boston terrier	16	Foxhound	3
Boxer	15	Cairn terrier	3
Farm shepherd	15	Bulldog	3
German shepherd	14	Welch terrier	2
Collie	12	Airedale	2
English setter	12	Pointer	2
Smooth fox terrier	11	Papillon	2
Dachshund	10	Keeshond	1
Springer spaniel	6	Staffordshire terrier	1
Wirehaired fox terrier	6	Bull terrier	1
Spitz	6	Retriever	1
Beagle	5	Golden retriever	1
Pomeranian	5	Chow	1
Great Dane	5	Sealyham terrier	1
St. Bernard	4	Pekingese	1
Scottish terrier	4	Kerry blue terrier	1

Despite the small numbers involved, Table II tells an interesting story of the breed incidence of encephalitis. Only those breeds where ten or more representatives appear in the foregoing list are included because any fewer might have no significance.

TABLE II—BREED INCIDENCE OF ENCEPHALITIS

Breed	Number of Dogs	Number Developing Encephalitis	Percentage
Cocker spaniel	70	14	20
Mongrel	40	10	25
Irish setter	17	3	17
Boston terrier	16	2	12
Farm shepherd	15	4	26
German shepherd	14	6	42
Collie	12	3	25
English setter	12	2	16
Smooth fox terrier	11	2	18
Dachshund	10	3	33

One conclusion seems quite obvious—dogs with broad heads and short faces (brachycephalic) are markedly less susceptible to the development of encephalitis following housedog disease than those with long, pointed noses like German shepherds, farm shepherds, dachshunds and collies (dolichocephalic).

Of the 15 boxers presented for diagnosis all recovered uneventfully. So did 14 of 16 Boston terriers; whereas six of 14 German shepherds succumbed.

The notion commonly held that the mongrel dog is far more hardy than the purebred meets with little support in this study. Twenty-five per cent of the mongrels died—a much higher percentage than for many pure breeds.

It is well to mention again that this study included more than 800 dogs, all of which had definite symptoms of housedog disease. The 309 complete records tell only part of the story. Among the 800 the same trends held with one exception. Only three of 17 Irish setters developed encephalitis among the 309 complete histories but the percentage is much higher in the larger series. It is the impression of all of us here at the clinic that the type most likely to develop encephalitis is the small farm shepherd and the German shepherd puppy at teething time. In most of these dogs the proportion is very unfavorable. In contrast not one English bulldog or boxer type has so far developed encephalitis although at least 50 of the two breeds have been observed with the primary (housedog) disease.

Treatment

No worthwhile treatment has been discovered for housedog disease nor for the encephalitis that so often follows it. About the time treatment is commenced the disease is generally starting to subside. Such seems to be the case with the common cold in man, which housedog disease so much resembles. For just two of the three commonest sequels (swollen tonsils and pneumonia) are there remedial measures of value.

Tonsils generally retract into the clefts after a week or two but when they remain enlarged, tonsillectomy may be resorted to. Painting the tonsils with iodine, tannic acid and glycerine affords some relief, or *laissez faire* may appeal to the client who is willing to put up with some coughing on the part of his pet, in the hope that the tonsils will eventually retrogress to the point where they no longer annoy the dog. In such cases enlarged tonsils have caused repeated spells of coughing, and shivering, with some elevation of temperature. Removal has prevented future attacks. But removal of the tonsils does not prevent encephalitis.

Pneumonia which follows housedog disease is a bacterial infection, probably attacking the weakened tissue *via* the upper respiratory tract. During the winter of 1941-1942, just 40 cases of pneumonia followed the disease. During the past winter sulfathiazole was given to all dogs having the primary disease and no case of pneumonia developed. Many dogs had pneumonia as a complication of housedog disease at the time they were presented for treatment. Most of these cases were cured by the administration of sulfathiazole or sulfadiazine.

Encephalitis is another matter. Fifty-eight dogs died of it. As has been said, puppies most often develop it. Some dogs have only slight attacks of the disease and, if kept quiet and nursed carefully, recover. Nembutal is useful in such cases to keep the patient quiet and free from fits which weaken him. Some benefit appears to have been derived from this treatment in severer cases but usually hope turns to despair before many days; the disease continues to

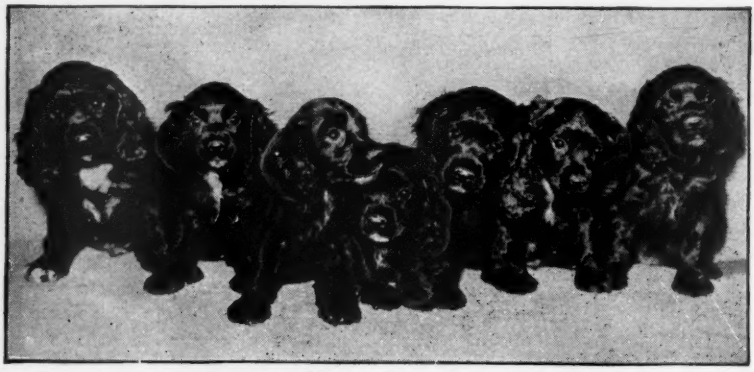
develop despite the fact that the encephalitis symptoms are repressed.

Vitamin concentrates have been used in large doses. First thiamin chloride was given orally and hypodermically, with no benefit whatever. Then large doses of injectable B complex were given. In several cases real benefit appeared to ensue but when it was tried critically with untreated controls, there was no difference; the untreated controls did as well as the treated dogs. It must be remembered that during the encephalitis dogs generally eat well except in the last stages, so large amounts of vitamins may be fed. This was tried too, giving yeast and concentrates, all to no avail.

Finally a study was reported showing benefits from injecting vitamin C in the chorea cases[6]. A large amount of the vitamin was obtained and injected (50mg. per dog per day for 7 to 10 days)—again to no effect. Dogs suffering from chorea, if allowed to live, are still suffering from it. In Doctor Zimmerman's descriptions of brains of affected dogs, most of which are from dogs in this study, the extreme damage from the disease will be noted. How can this damage be corrected? More than $100 worth of vitamin concentrates were used but the expenditure was wasted.

Sterile milk, omnadin, many sulfa drugs, sodium iodide, iron cacodylate, typhoid serum all have been carefully tested and none relieved the encephalitis.

One treatment entirely empirical and wholly impractical seemed to give good results. In five of the author's own dogs inducing abscess formation was tried. Two recovered permanently. In the others the encephalitis appeared to be checked only to recur about a week after the abscesses had ruptured. These abscesses were made in the shoulder muscles using 5cc of a yeast emulsion. The whole shoulder and leg swells, and when the abscess ruptures the encephalitis symptoms, if taken at the first sign of the disease, appear to cease.



Cocker spaniels are the most popular house dogs and, as is to be expected, they lead all other breeds in the number of cases of housedog disease. Puppies, such as these, are usually vaccinated against distemper, probably by the Laidlaw-Dunkin method—the explanation as to why it may not be a protection in all diseases of the distemper group will be found in this article

This treatment was tried in the early stages on seven cases of Carré distemper five years ago with excellent results.

Serum was tried in the treatment of encephalitis and appears to offer promise if made from a dog that had recovered from the disease and if used in large amounts. Ten and 20cc doses in medium-sized dogs proved worthless but 100cc doses in 40-pound dogs showed promise. Five dogs appear to have been cured by it. Each was chosen at the first fit or evidence of fright or intense nervousness. The improvement was rapid and permanent. But large doses in dogs which have had fits for several days was wasted serum.

All the serum obtained from one large dog was used for four small dogs mentioned above and all recovered. One poodle had a mild twitch in the head. Within two days

[6] Egan, F. D., 1942. Vitamin C in canine practice. *M. S. C. Vet.*, 11:4, pp. 117-118.

the twitch was gone and the dog grew well and has remained healthy.

Such serum is easily made by drawing the blood *via* the jugular vein of an anesthetized dog, into sterile containers, allowing it to clot and chilling it, then pipetting the serum into rubber stoppered containers for use. This is preserved with merthiolate 1 to 10,000, or with some other satisfactory preservative. When used fresh, the results mentioned followed.

Serum made from dogs that had recovered from Carré distemper was no value against encephalitis following housedog disease, even when used in large doses (2cc per pound of body weight). Large doses of commercial hyperimmune anticanine distemper serum were equally ineffective against housedog disease.

But convalescent serum made from dogs recovered from encephalitis, appears somewhat meritorious if injected in dogs in the early pharyngo - laryngo - trachitic stage. The normal expectancy of puppies with this disease to develop encephalitis was 35% in our series. When doses of 20cc of this serum—all from the same dog—were injected into 20 dogs, all under one year old, not one was returned with encephalitis. This seems more than a series of coincidences.

Another dog, a year after it had recovered from encephalitis, was sacrificed and a serum was made from the blood. As a comparison with the first dog's serum another 20 dogs were injected with 20cc each. In this series, little benefit was observed from the injections; four dogs, which was somewhere near the normal expectancy, developed encephalitis.

Serum was made from the blood of another dog in the convalescent stage and eight young dogs were injected with 20cc each. One of these developed encephalitis. All of which may indicate that serum can be made which is of some value, but how much, remains to be determined.

Experiments With Sulfanilamide on Dogs

A number of studies have been made of the effect of sulfanilamide on the acid-base equilibrium of man and animals. The gen-

eral result of these studies have indicated that in some patients and animals evidence of acidosis is present following sulfanilamide ingestion, and the use of an alkali with the drug has been suggested and widely followed.

In an experiment by Free, Bowman and Davies[1] in which female dogs were employed, this supposed tendency of sulfanilamide to effect the acid-base equilibrium was not supported by the findings.

The serum pH was not appreciably altered from the control level by sulfanilamide, sulfanilamide and sodium citrate or by sodium citrate alone. The serumchloride was maintained at approximately control levels in all experiments. The hemoglobin content of the blood was not significantly altered. The erythrocyte and leucocyte counts of the blood were not appreciably changed and no significant changes in the total base, protein phosphate or undetermined acid were noted between the beginning and the end of the experiment.

[1] Free, Alfred H., Donald E. Bowman and Dean F. Davies, 1943. The effect of continued sulfanilamide ingestion on the acid-base equilibrium of the dog. *Jnl. Lab. & Clin. Diag.* 28:9, pp. 1109-17.

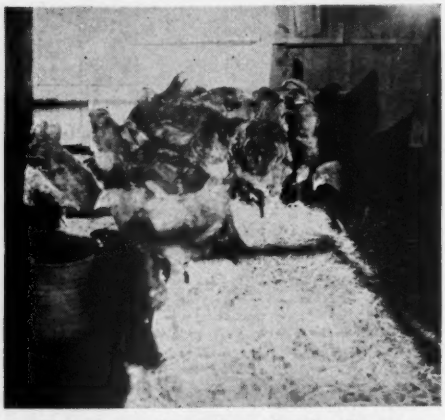
Erysipelothrix rhusiopathiae and Pasteurella avicida in Chickens

By FRANK BREED
Lincoln, Nebraska

OCTOBER 9, 1942, a shipment of chicken specimens was received for bacteriological examination from Dr. J. L. Hoylman, Loup City, Nebraska. The specimens consisted of the heart, spleen and digestive tract.

The history, which was very meager, contained the information that apparently this was an atypical outbreak of fowl cholera, or of some disease with which they were not familiar. The younger birds did not respond to the form of treatment previously used on the older birds on these premises.

The gross pathology consisted of severe hemorrhage of the epicardium, enlargement of the spleen and hemorrhages scattered throughout the mucosa and, to some extent, the serous coat of the digestive tract.



The history seemed to indicate that the disease was apparently an atypical outbreak of fowl cholera

Cultures were prepared from each of the tissues received on glycerine agar, sugar-free broth, plain broth and broth containing dextrose, lactose and saccharose. Growth was present in all the tubes at 24 hours. There was no gas in the media containing the sugars. In the broth, both sugar-free and plain, there was a uniform clouding throughout the media and a silky sheen, which is quite characteristic of *Erysipelothrix rhusiopathiae*, appeared during the first 24 to 30 hours. Microscopic examination of both the agar and broth cultures showed the presence of many typical gram-positive rods, slender and slightly curved. Such rods are characteristic of the erysipelas organism. Gram-negative rods, showing the typical bipolar staining, were also present in moderate numbers. Microscopically, the organisms were so characteristic of *E. rhusiopathiae* and fowl cholera that our findings were reported immediately to Dr. Hoylman and the use of anti-erysipelas serum in the control of this infection was suggested.

Further culturing by means of dilution plates revealed that the gram-positive, slender and slightly curved rods were far in the majority; the plate cultures also showed the presence of colonies which were different in size and character. Colonies were picked and pure cultures of the two organisms were obtained.

The cultures containing the suspected gram-positive rods were injected in the breast muscles of two pigeons both of which were dead in approximately 65 hours. Microscopic examination of the heart, blood and liver showed the same typical gram-positive rods in large quantities. Cultures from the organs of the two pigeons resulted in the recovery of the organism in pure form. Upon further study the gram-positive rods were identified as *E. rhusiopathiae* and the gram-negative, bipolar staining organisms as *Pasteurella avicida.*

The swine erysipelas organism has been found either as a saprophite or as a pathogen in many species other than swine. Van Es, in a revised edition (June, 1942) of Research Bulletin 84, Nebraska Experiment Station, states "Reports are available pertaining to their presence in man, cattle, dog, duck, fowl, turkey, pigeon, mud hen, parrot and the birds of a zoological garden." In the same bulletin (page 8) reference is made relative to the investigations

of Meloni, in which it is pointed out that old geese, ducks and common fowls could not be infected with severe erysipelas but that inoculations were successful when young birds were selected.

The birds infected in this natural outbreak were approximately six months of age. The older birds were not affected. This tends to confirm the experience reported by Meloni in his investigation. It is possible that the pasteurella organism may have played a part in the natural infection with the erysipelas organism by causing a lowered resistance, especially in the first group of birds affected.

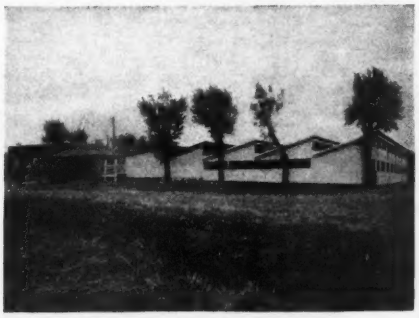
The following history and information has been furnished by Dr. Hoylman for the completion of this report.

"The flock consisted of about three hundred birds. Losses started when birds were found dead under the roost or on the ground. The histories were very suggestive of fowl cholera, a disease which had previously existed in the older birds on this farm. This outbreak of cholera in the older birds responded very satisfactorily to medicated water, sanitation and some change of environment.

"When the losses started in the younger birds it was thought to be cholera and, without seeing the flock or holding autopsies, the same method of handling was instituted and continued for about three weeks. As the losses were so heavy a visit to the premises was made. During this period about 100 birds had died and the losses were increasing. One peculiar thing which made me feel this might not be cholera was, in the early stages the affected birds were very excitable and difficult to catch. As the disease progressed the sick birds would huddle by themselves as though they were trying to keep warm. The feathers were ruffled and the birds were inclined to keep off their feet. Sudden deaths were not occurring at the time. Some of the sick birds lived nearly two weeks. It was noted that the birds which were slightly off were badly soiled around the vent and had lost flesh very rapidly. On autopsy, a considerable quantity of fluid was found in the abdominal cavity with a severe enteritis extending all through the bowels of some of the birds. The contents were more or less watery, containing a yellowish or creamy material. The heart was soft and flabby and the epicardium contained a goodly quantity of petechia.

"By the time your report of the finding of erysipelas and cholera organisms was received, about 200 of the birds had died.

"The treatment used was 2cc anti-erysipelas serum per bird. Most of the remaining 100 birds appeared to be a little off at the time of treatment. Losses stopped in this group immediately and no further losses occurred. There were six very sick birds, to which we gave the serum; these birds had been sick from one and two weeks. They all died. The serum seemed to have no beneficial effect.



Birds were found dead under the roost or on the ground. By the time diagnosis was made and treatment could be begun, 200 of a flock of 300 young chicks had died

"The owner has just reported (after a period of about 10 months) that no further losses have occurred, that the remaining flock has done well and are still laying."

This report has been given very much in detail because it is felt that this same condition might be encountered in flocks of young fowls on other farms in the middle west where erysipelas in swine has become prevalent. Further, there is a possibility that the adult fowl might be a reservoir for the spread of erysipelas to areas where the disease as yet does not exist.

This report would not have been possible without the whole-hearted cooperation which Doctor Hoylman has so generously given.

Autobiography of a Veterinary Practitioner

By E. T. BAKER, Moscow, Idaho

XX
Everyday Problems

IN MY work I have always found the everyday problems more important than those less frequently seen. For this reason, I have often wondered, when attending association clinics, why so much time is spent on operations seldom performed in general practice.

I saw one entire afternoon devoted to amputating an udder from a cow in which all the hemostats in that part of the country were required. While the operation was apparently a surgical success the patient died within several hours. Now, I have never had occasion to perform this operation because it did not seem practicable for field surgery. I often see gangrenous quarters that have "busted" according to the owner, and the entire part had sloughed out. By treating this as an open wound, and supporting the patient with good food, vaccines, antiseptics and blood tonics, she usually lives.

How much more interesting and instructive would have been various methods of restraint, or to see a "spider" removed in a skilful and deft manner. Even an operation of rumenotomy, performed under field conditions, would be helpful.

It would have been more interesting if some one had demonstrated various methods of restraint—for example, the one man casting harness illustrated above in which a two-year-old colt is restrained

Public Demonstrations

General practitioners soon realize they cannot be wizards in all lines of veterinary work. Some of us develop skills along certain kinds of practice, depending upon the types of animals handled. Place me among a bunch of Kentucky thoroughbred horses and I would be lost, for I have not treated a dozen cases of light saddle or harness horses in 20 years. Throw me among a band of sick sheep and I have a faint idea of what to do and how to diagnose the trouble.

Watch two men attempt to give a drench, a capsule or even a hypodermic injection. One man possesses the "knack" of working easily while the other makes the hardest kind of work out of the job.

One reason why many proficient practitioners dislike to perform in public is that they know there are failures in even the most simple procedures. In our practice we give many capsules to horses and cattle every month and rarely miss a "shot," but occasionally we run across an animal that is too smart to swallow. It calmly allows the capsule to roll out of its mouth, and dares us to try again. This time we take more pains in handling the "critter" and are usually successful the second time.

From the viewpoint of the owner and bystanders, a veterinarian's skill as a practitioner is largely judged by the manner in

which he handles the patient and administers the medicine. Consequently we welcome demonstrations of this nature, and

When two men attempt to drench a sheep its head should not be raised very much above the level of its back and its nose must not be extended, entailing difficulty in swallowing. Suffocation from the liquid going to the lungs is the penalty for disregarding this admonition

have picked up many useful pointers as a result.

One little wrinkle I invented myself simplifies the immunization of puppies. Having no small animal hospital, I do the work in my medicine room with the owner restraining the animal. A three-inch length of common intravenous tubing is attached to the end of an eye dropper, and before the puppy has even time to get scared he gets this full of elixir lactated pepsin. Then, while he is licking his chops, the immunizing injection is given and he hardly notices it. If this dose of pepsin is repeated just as the patient is leaving it will look forward to a return visit and more of that good tasting medicine.

Another thing, when I leave some medicine to be given as an electuary to a sow "down behind," or getting over eclampsia, I always suggest the client use his left hand. Then, if he happens to get a finger or two chewed off he will not miss them so much. Of course, the routine is reversed for a southpaw.

When a client brings in a cat for treatment, I always ask him to hold the patient. Then, if anyone is scratched, it is not I.

Also, it would be interesting to see how an intravenous injection could be given to a wild case of azoturia wedged in between a barbwire fence and a thorn bush.

Mother Experience Good Teacher

At one clinic a very young and very confident practitioner attempted to demonstrate his method of administering worm capsules to swine. While one can gain considerable dexterity in this art by experience, in my own limited hog practice I prefer to give the "dope" in the feed.

However, our young expert was "rarin' " to go and, with the sleeves of his immaculate white gown rolled up, he waved for the minions to bring in the porcine victim. This was done, and the virtuoso proceeded to administer the capsule with all the grace of a hired man reaching for his fourth helping of fried chicken. But something

The client is always allowed to hold his cat so if any scratching is done I won't be hurt

seemed to go wrong. There was a slight gasp; then all was silent, and the curl began to disappear, as the animal quietly breathed its last.

Undaunted, our hero waved for another porker, and it, too, met a similar fate. We

felt sorry for the young fellow because, ordinarily, he could probably do the work with very little trouble but this happened to be before a crowd when everything seems to go wrong. It was much like the man who strenuously objected to being hanged on a Monday. He said it was a lousy way in which to start the week.

Signs and Superstitions

Regardless of what we may think of "signs of the moon," many of our clients have great faith in them. They insist on having certain operations performed when the "sign is right," and the practitioner who pooh-poohs this idea will not influence people or make friends.

In order to cater to my clients who go by the moon, I mark each day of the month with its appropriate sign. For example, July, 1943, ran like this: 1st, 2nd and 3rd, Breast; 4th and 5th, Heart; 6th, 7th and 8th, Bowels; 9th and 10th, Reins; 11th and 12th, Loins; 13th and 14th, Thighs; 15th and 16th, Knees; 17th and 18th, Legs; 19th and 20th, Feet; 21st, 22nd and 23rd, Head; 24th and 25th, Neck; 26th and 27th, Arms; 28th, 29th and 30th, Breast; 31st, Heart.

These sign experts assure me that an animal operated on in the sign of Head will swell up greatly and fail to heal properly. If done in the sign of the Heart fatal bleeding may take place, and blood poisoning is sure to follow work done in the sign of the Bowels. The proper time, they aver, is "from the Knees to the Feet." At that sign of the moon, all the swelling quickly goes down into the ground, and the patient makes a rapid and complete recovery. Why should I argue about it? I'm not the one the school board hired to do the educating in this community.

I had a client, engaged extensively in horse raising, who claimed if a colt was weaned in the sign of the breast, neither colt or more would notice the process. He claimed even the mare's udder would dry up naturally.

When Sam Johnson came in to have some good white faced calves castrated, he began:

"Kin you do them calves around the 20th, Doc? That's when th' sign is jake. I ain't never had a calf even swell up when you done it then."

We drive out to Sam's ranch early on the 20th, and he has the animals quiet and cool in a shed. As we get ready to leave, he pulls out his checkbook and pays the fee with a smile. He is perfectly satisfied because we have performed the work when he wanted it done. The calves all do well, and he has great faith in both our ability and the sign.

I am mentioning this subject because I had to learn the hard way. I lost several good clients for belittling their belief in signs. Then, one day in a rational moment, which comes to all practitioners at irregular intervals, I looked the question square in the face. It has paid good dividends ever since.

Many capable practitioners dislike to perform in public because they know that you never can tell when things will go wrong

(To be continued)

★ ★ ★ BUY WAR STAMPS ★ ★ ★
AND BONDS

Ergot Poisoning in Cattle

Ergot is a fungus which affects and finally replaces the seed of cereal grasses; especially is this true of the sclerotium of *Claviceps pururea*, which replaces the grain of the common rye.

Above is shown one of the cows in which the left hind foot has sloughed off and its mate is badly injured

Acute fatal poisoning by ergot is rather rare in animals but the chronic type is more common. It is caused by taking large amounts into the digestive tract at one time or by repeated smaller amounts of the substance.

A typical case of chronic ergot poisoning occurred in Nemaha County, Kansas. Of a herd of 30 head of cattle, 12 were affected by gangrenous sloughing of the feet—chronic ergot poisoning.

In the bloodsteam the active principles of ergot constrict the arterioles so extensively that the circulation is almost completely cut off. The gangrene affects those parts poorest in blood supply, especially the extremities. Lesions vary from simple sores around the top of the hoof and in the interdigits to loosening of the hoof or sloughing of an entire limb. A circular line of demarcation appears between the healthy and gangrenous tissue.

In this case ergot poisoning was suspected. The rye hay on which the cattle had been fed was examined and ergot was found to confirm the diagnosis.

The remedy for this condition depends upon the proper diagnosis and removal of the source of the ergot. Healing may be speeded up if the gangrenous parts are removed surgically.

RAYMOND H. COOK

Courtland, Kans.

Canine Pseudo-Hermaphrodite

A four-months-old collie was presented at the veterinary clinic of the Kansas State College in May, 1943, to be spayed.

The dog was secured in dorsal recumbency on the operating table, and the ventral abdominal area shaved and disinfected with tincture of iodine, when a slight abnormality of the vulva was noted. It was situated about 1½ inches anterior to the normal position. Palpation revealed a rudimentary penis, ¼ inch in diameter, at the dorsal (posterior), commissure which could be followed backward and upward over the ischial arch. By means of a flexible probe the urethra was located and followed over the ischial arch also.

Palpation and inspection failed to reveal a scrotum or testicles, so exploration of the abdominal cavity for the uterus and ovaries was decided upon. The female organs were not found, nor were retained testicles located.

The owner was advised that a search would be made for rudimentary testicles located subcutaneously if he cared to bring the dog back in one week.

This case emphasizes the desirability of a careful pre-operative examination of the patient. A casual inspection would have revealed nothing abnormal since the external appearance of the vulva was normal and some trauma of the abdominal viscera might have resulted from a fruitless search for the female genital organs.

KENNETH ZIMMERMAN

Coffeyville, Kans.

Nails Ingested by Young Chicks

There is very little in the literature concerning foreign bodies, such as nails and other sharp objects, in the digestive tract of chickens. There is, however, a short discussion of the surgical removal of foreign objects from the crop of fowl in the new work, "Diseases of Poultry," by Beister and Devries. The following cases will show this condition is of some economic importance and not just a curiosity.

Five dead two-weeks-old chickens were presented with a history that, in a flock of 300, 14 were found dead that morning and 11 the morning before. No symptoms were noticed by the owner before the chickens were found dead. Upon routine post-mortem examination, galvanized roofing nails were found in the gizzard and crop of two and in the gizzards of the other three. Two nails were found in the gizzard of one. The owner, who was watching the necropsy, was much surprised to see the nails but recalled that he had a can of such nails in the brooder house while repairing it.

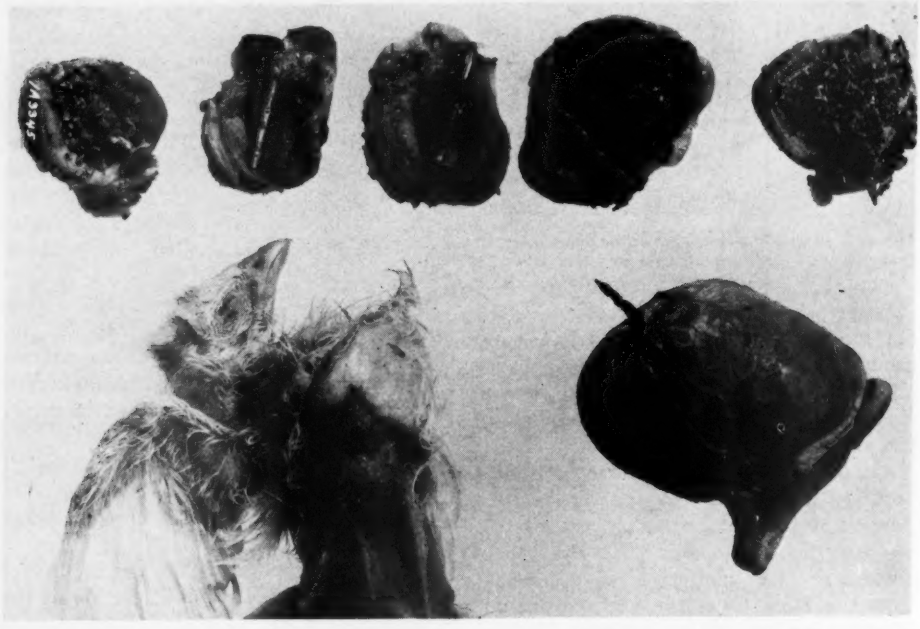
It seems mechanically impossible for a chick less than two weeks of age to swallow a nail one inch long with a flat head five-sixteenths of an inch in width.

Later a chick, three weeks of age, which had a roofing nail in its gizzard, was presented. Another owner, who made her own necropsies, reported finding roofing nails in the digestive tracts of 15 chicks that died out of a flock of 200.

L. D. Bushnell, director of the poultry disease laboratory, Kansas State College, tells of a farmer who brought in a fairly large number of young chickens which he was sure had been poisoned. On necropsy it was found they had all swallowed large headed, two pronged, upholstering tacks which had been dropped on the ground while the farmer was reupholstering his automobile a few days before.

Death is occasionally caused in mature chickens by a nail perforating the muscular wall of the gizzard.

While foreign objects in the digestive tract of fowl is not a cause of large losses,



Nails should not be left carelessly about a chicken yard as even chicks two weeks of age have swallowed nails an inch long

it occurs frequently enough to add another reason for making post-mortem examinations on all sick or dead chickens presented for diagnosis. As in many other conditions in poultry, the symptoms and history are in no way pathognomonic.

S. LESTER JACKSON

Parker, Kans.

Parturient Paresis Remote from Parturition

Last July I was called to see a fine, big, grade Holstein cow. She was down in the pasture and had been unable to rise for several hours; she was about seven months along with calf. I gave her almost everything in the pharmacopoeia but the title page; but it did no good. She consistently refused to get on her feet for three days and nights. We rigged up a block and tackle and forcibly lifted her up, but she refused to stand. She always lay in a natural position and would drink and eat when food and water was offered.

As it was near my home, I made several calls. She seemed to be all right except she persisted in remaining in a recumbent position. My client was a well-to-do "city farmer" (if there is such a thing) and every time I called I was surrounded by a considerable audience of the neighbors who would look at me and then at the cow and then silently shake their heads in unison.

I spent most of a day digging up old journals in an effort to find something referring to such a case. Finally, in a back number of VETERINARY MEDICINE I found an article that gave me a lead. So, late that evening, I inflated her udder and gave 40cc. of a saturated solution of magnesium sulphate subcutaneously. I noticed, as usual, the head shaking of neighbors grouped about. Then I asked my client if he ever walked in his sleep. Upon his puzzled reply that he could if it was necessary, I told him that if he would get up between one and two o'clock the next morning, and go to the pasture, that he would meet his cow coming to the barn.

The next morning, just at daylight, my client called to tell me that the cow was up grazing with the other cattle. The wise neighbors concluded that "it must have been that shot that Doc gave her," as "who ever heard of pumping up a dry cow."

The cow made an uneventful recovery and delivered a fine calf at full time. I believe this case was either acetonemia or grass tetany, or possibly, a combination of the two.

A statement in the April issue of VETERINARY MEDICINE by Leroy E. Bowen, "the etiology of complicated milk fever is still wrapped in mystery," is 100% true so far as I'm concerned.

REMBRANDT MORGAN

Winfield, W. Va.

Castration of Rabbits*

The advantages of castration of rabbits are that the owner of the commercial rabbitry can keep and fatten large numbers of females and castrated males in the same pens without the segregation which would necessarily have been required if the males had not been castrated. Also, the gain, per pound of feed consumed, is greater in the castrated males than in those not castrated. It is said that the meat of castrated rabbits is more palatable.

In laboratories and market houses where large numbers of rabbits are kept it is an advantage to keep the rabbits together, which would not be possible were the males not castrated, as they would attack the females and render them unfit for market from the buyer's viewpoint. Where the number of rabbits raised in a season exceeds 100, the economies from castration of the young males are considerable. However, since the value of the individual rabbit is low, the practitioner must needs plan the work so that the operation can be performed for a small fee.

It has been found that the most desirable operable age is two months, although the animal may be operated successfully at any age.

The castration of rabbits has not been practiced extensively in the past, due to the high mortality experienced by some operators. Statistics show that approximately 50% of the operations result in death from prolapse of the intestines through the operative incision.

*Reprinted on request from *Vet. Med.*, 28:6, 1933.

In this operation a knowledge of the anatomy of the male genital organs involved is essential in order to undertake and complete the operative procedure required.

The scrotal sacs are located one on each side of the penis just lateral and anterior to the preputial opening. After incising the scrotal skin, the first tissue encountered is a thin layer of muscle which forms the outer layer of the sac of the testis. This, the cremaster muscle, is continued in part with the internal oblique muscle of the abdominal wall, and also with fibers from the transverse abdominal muscle. The next tissue is the tunica vaginalis propria, the parietal layer of peritoneum, lying directly beneath the cremaster muscle which is continuous with the peritoneum of the abdominal wall forming the internal lining of the scrotum.

Because of the peculiar anatomical arrangement of the abdominal muscles forming only an inguinal opening and not the usual inguinal canal, the testes pass freely from the peritoneal cavity to the scrotum, this depending on the contraction and retraction of the cremaster muscle.

The next tissue encountered is a layer of the tunica vaginalis propria which is closely adherent to the testicle.

The testes are attached to their respective sacs by short cords containing smooth muscle fibers, the gubernacula testes.

As the testes commune freely with the peritoneal cavity they are usually found either just within the cavity or just outside the body wall, depending upon the tensity of the cremaster muscle. The testes are seldom found within the scrotum proper, except at breeding time. For this reason the rabbit must be properly restrained before castration is attempted.

The assistant places himself upon a chair or high stool. The rabbit is turned upon its back with the operative area toward the operator, who stands directly in front of and facing the assistant. (The assistant wears gauntlet gloves, to prevent being bitten as an anesthetic would be impracticable.) Resting the back of the rabbit on his knees, the assistant holds the legs by placing the forefinger and thumb around the foreleg and the other three fingers around the rear limb of the same side of the rabbit. This is repeated on the other side. By grasping the animal in this manner, the legs are held apart laterally, and the back of the animal arched. The operator clips the hair and paints the field of operation with tincture of iodine.

Place the thumb and the first two fingers on the periphery of the inguinal ring and push gently downward, at the same time gradually closing the fingers. Usually they will enclose the testicle.

Gently and carefully pull the testicle toward the scrotum. After the testicle is within the scrotum proper, force the scrotum and testicle as far posterior as possible, which is just lateral to the anus.

With a sharp scalpel incise the extreme posterior end of the scrotum, the line of the incision being perpendicular to the long axis of the body.

Make the incision just large enough for the testicle to pass. Repeat this incision in the tunica vaginalis propria, cutting all underlying tissue until the testicle proper is exposed. Grasp the testicle with the left hand, and draw it backward until the cord is well exposed. Use an emasculator in severing the cord or scrape through it with the scalpel as close to the epididymis as possible.

Take the scrotum between the forefinger and thumb and pull it backward until the stump of the cord is retracted within the scrotum. This procedure is to be repeated with the other testicle.

The reason for arching the animal's back, in the manner first mentioned, is to have the incision of the tunica albuginea and inguinal ring at different places when the animal is in a normal position, thereby preventing intestinal prolapse.

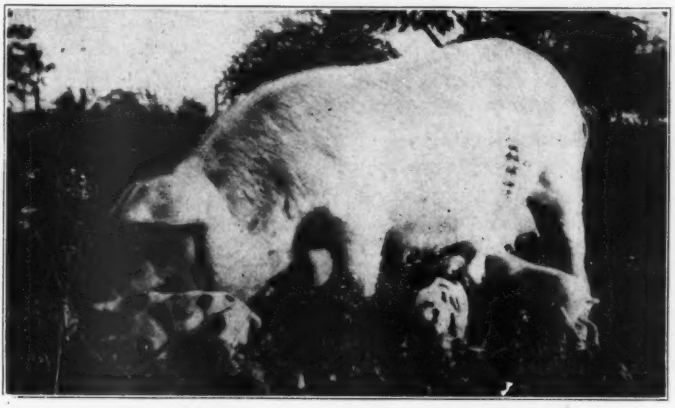
It is thought that improper restraint of the animal, too large an incision in the tunica albuginea, and not drawing the testicle backward far enough have been the difficulties of most individuals in castrating rabbits.

G. S. McKee.

Columbia, S. C.

Dystocia and Cesarean Section in a Sow

On an afternoon in April, 1943, a sow was presented to the veterinary clinic of the Kansas State College with a history of having been in labor since the day before. It was her first parturition and she had delivered one pig normally at the beginning of labor. She was down and showed paresis of the hind legs and, upon examination, a fetus could be felt in the uterus. The birth canal was lubricated with mineral oil and the sow given ½cc of pituitrin intramuscularly. In about an hour she was examined again and two dead fetuses removed by traction. A third fetus could be felt but could not be removed. The birth canal was again lubricated and another dose of pituitrin given. The sow was examined at frequent intervals but the fetus seemingly could not be removed. Pituitrin was administered again but to no avail.



The sow recovered after the cesarean section and was sent home 12 days after she was brought to the clinic

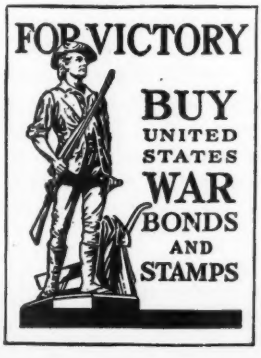
Although it is usually not advisable to perform a cesarean section if a sow has labored for more than 24 hours, it seemed the only solution in this case.

The region in the right flank was shaved, cleaned and tincture of iodine applied to the area. Anesthesia was accomplished with chloral hydrate solution per rectum (12:5g of chloral hydrate in 250cc of water) and by infiltrating the operative area with 1½% solution of procaine hydrochloride. A vertical incision (8 to 10 inches in length) was made in the skin, starting just below the transverse processes of the lumbar vertebræ and halfway between the last rib and the external angle of the ilium. The muscles and peritoneum were incised and upon reaching into the preitoneal cavity the horn containing the fetus was located and brought through the opening. An incision was made through the uterus and over the fetus large enough to permit its removal. The uterus was then sutured with catgut, using a double row of Lembert sutures, washed with sterile physiological saline and returned to the peritoneal cavity. The peritoneum and muscles were closed with a continuous suture of catgut and the skin with interrupted sutures of umbilical tape.

The operative area was sprayed daily with camphenol antiseptic. The sow soon regained the use of her hind legs and 12 days after she was brought to the clinic was sent home.

JOHN HOINS.

Leavenworth, Kansas.



Abstracts

Brucellosis in Man

That the chronic phase of brucellosis in man is often overlooked, is the belief of D. L. Urschel.[46] In 124 cases with suggestive histories of brucellosis he found (testing by means of intradermal vaccine), that 70, or 56.4% gave a positive skin reaction to *Brucella abortus* and *Brucella suis* vaccine —but just 53 of the cases had what could be considered clinically active brucellosis. Of these 23% gave a history of exposure to brucellosis as they all consumed raw milk.

Oral Lesions in B_1 Avitaminotic Dogs

In studies conducted at Vanderbilt University, Govier and Greig had occasion to produce avitaminosis B_1 in dogs.[41]

This was done in two ways: first by the feeding of the diet suggested by Goodsell which contains casein, sucrose, cottonseed oil, agar and cod liver oil, with autoclaved brewer's yeast to supply the other B complex vitamins, and second, by supplying the diet of Schaefer, McKibbin and Elvehjem, in which, instead of brewer's yeast, riboflavin, nicotinic acid, pantothenic acid, pyridoxine and choline, in adequate amounts, was administered by stomach tube to the dogs twice weekly. Many of the animals in both groups developed necrotic erosions beginning around the teeth and sometimes becoming so severe as to extend almost entirely around the lower jaw. Most of these lesions showed a thin mucopurulent discharge and a mildly fetid odor. The authors had supposed that these lesions, when they occurred in animals on the diet of Goodsell, were produced by the failure of the animals to eat the B complex vitamins contained in the autoclaved yeast, since it is well known that B_1 avitaminotic animals have little appetite. However, when, with the diet of Schaefer, *et al.*, B complex vitamins were supplied by stomach tube, the lesions still occurred.

Athough it is known that dogs normally synthesize their own vitamin C, on account of work of Sure, *et al.*, they surmised that perhaps, under the experimental conditions named, they were unable to do so. They showed that various members of the vitamin B complex, including thiamin, were necessary for synthesis of vitamin C in the rat, another animal normally synthesizing its own ascorbic acid.

On this basis all the dogs were given 10mg of ascorbic acid twice weekly with the other vitamins by stomach tube. Those animals showing the oral lesions promptly healed, and no more lesions appeared in the other dogs.

Prevent Shock and Death

Ten dogs were anesthetized with pentobarbital sodium by Fell and Hanselman[45] and kept under light anesthesia for about 12 hours. For 20 minutes the right-hand extremities to the upper third of the thigh were immersed in a mixture of solidified carbon dioxide and 95% alcohol at 55°C. Within two minutes the immersed portion was frozen. Of these animals, five received no treatment but the legs were allowed to thaw out at room temperature—all of these developed clinical shock and four of them died within 13 hours.

The frozen limbs of the other five dogs were wrapped in several layers of sheet wadding and these covered with a plaster cast. All of these dogs lived. The plaster cast afforded a firm even pressure and allowed the encased legs to thaw out quite slowly. However, in four of these five treated dogs the legs became necrotic. Within 48 hours the necrotic limbs of one dog were amputated and within 8 to 11 days those of two others were removed—all three animals made uneventful recoveries. The authors conclude that it is desirable to apply pressure dressing to frozen, compressed or burned parts—this should be done before swelling sets in, immediately upon release from compression, after a burn or before the frozen part has begun to thaw.

[46] Urshel, D. L., 1943. Brucellosis—a report of 53 cases with an introductory on intradermal vaccine therapy. *Ind. St. Med. Assn.*, Jul., 36, p. 294.

[41] Govier, Wm. M., and Margaret E. Greig, 1943. Prevention of oral lesions in B, avitaminotic dogs. *Science*, 98:2540.

[45] Fell, E. H., and R. Hanselman, 1943. Prevention of shock and death by immediate application of pressure dressing to severely frozen limbs of dogs. *Ann. of Surg.*, 117, p. 686.